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Pulse Wave Velocity and Vitamin D: A Narrative Review

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ABSTRACT

Pulse Wave Velocity (PWV) is a well-established, noninvasive biomarker of arterial stiffness and an independent predictor of cardiovascular morbidity and mortality. Emerging evidence suggests that vitamin D deficiency, prevalent worldwide, may adversely influence vascular function through mechanisms involving endothelial dysfunction, inflammation, and altered calcium homeostasis. However, the extent and consistency of this association remain under active investigation. This narrative review aims to critically examine the existing literature exploring the relationship between vitamin D status and arterial stiffness, as measured by PWV, and to elucidate potential physiological and clinical mechanisms underlying this interaction. A comprehensive literature search was performed using PubMed, Scopus, and Embase databases. Studies evaluating serum 25-hydroxyvitamin D [25(OH)D] levels in relation to PWV in adult populations were included. Both interventional and observational studies were reviewed, emphasizing methodological quality, confounding control, and population diversity. Most observational studies demonstrate an inverse association between serum 25(OH)D levels and PWV, suggesting that vitamin D deficiency is correlated with increased arterial stiffness. Interventional trials assessing vitamin D supplementation yield heterogeneous findings; improvements in PWV are more evident in vitamin D-deficient cohorts and those with elevated baseline cardiovascular risk. Mechanistically, vitamin D may modulate arterial stiffness through suppression of renin-angiotensin-aldosterone system activity, attenuation of vascular smooth muscle cell proliferation, and enhancement of nitric oxide bioavailability. Current evidence supports a biologically plausible link between low vitamin D status and increased PWV, though causality remains inconclusive due to inconsistent intervention outcomes and population heterogeneity. Further large-scale, randomized controlled trials with standardized vitamin D dosing regimens and longitudinal PWV assessments are warranted to establish therapeutic relevance. Understanding this relationship could refine cardiovascular risk stratification and inform preventive strategies targeting vascular health.

KEYWORDS: Pulse wave velocity, Vitamin D, Arterial stiffness, Cardiovascular risk, Endothelial function.

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INTRODUCTION

Vitamin D, recognized primarily for its role in bone and calcium metabolism, is a fat-soluble prohormone with extensive involvement in numerous physiological processes via interaction with vitamin D receptors [1]. Beyond its classical functions, a growing body of evidence highlights its critical involvement in cardiovascular health, modulating inflammatory responses, endothelial function, and vascular remodeling [2]. Deficiency in vitamin D has been frequently correlated with an elevated risk of cardiovascular diseases, encompassing conditions like hypertension, atherosclerosis, and myocardial infarction [3]. This narrative review aims to comprehensively explore the intricate relationship between vitamin D status and pulse wave velocity, a key indicator of arterial stiffness and an independent predictor of cardiovascular events. Specifically, this review will delve into the mechanistic insights explaining how vitamin D influences vascular function, critically appraise the epidemiological associations between vitamin D levels and PWV, and summarize findings from interventional studies examining the impact of vitamin D supplementation on arterial stiffness [1]. Furthermore, the review will consider potential discrepancies in research findings, examining factors such as patient populations, duration of intervention, and methodologies employed in assessing arterial stiffness, while also identifying critical gaps in the current literature to guide future research directions [4]. Particular attention will be paid to the bidirectional relationship between vitamin D and vascular pathologies, considering how deficiencies might exacerbate arterial stiffening and how improving vitamin D status could mitigate cardiovascular risk [1, 3].

VITAMIN D METABOLISM AND CARDIOVASCULAR HEALTH

Vitamin D deficiency is a pervasive global health concern, affecting a substantial proportion of the population, with estimates indicating that approximately 30% of children and 60% of adults worldwide experience insufficiency [5]. This widespread deficiency is particularly concerning given vitamin D's multifaceted roles in maintaining cardiovascular homeostasis, including the regulation of antioxidant mechanisms, cell proliferation, differentiation, and apoptosis [6]. Moreover, emerging research indicates that vitamin D modulates immune responses and inflammation, both of which are critical in the pathogenesis of cardiovascular disease.

PHYSIOLOGICAL ROLE OF VITAMIN D

Specifically, vitamin D can influence vasodilation by modulating vascular smooth muscle cell function and the bioavailability of vasodilators like nitric oxide, impacting vascular stiffness [7]. Furthermore, vitamin D has been implicated in the suppression of oxidative pathways and in regulating the sensitivity to the renin-angiotensin-aldosterone system, thereby exerting a protective effect against vascular remodeling [8]. Moreover, vitamin D receptors are expressed in various cardiovascular cell types, including vascular smooth muscle cells, endothelial cells, and cardiac myocytes, suggesting direct mechanisms through which vitamin D influences cardiovascular function [9]. In addition to these direct effects, vitamin D can also stimulate the expression of endogenous calcification inhibitors within the vascular wall, such as Klotho and Osteopontin, suggesting a role in mitigating arterial calcification [10]. The pathogenesis of arterial calcification is a tightly regulated process involving the phenotypic conversion of vascular smooth muscle cells into osteoblast-like cells, a process that vitamin D signaling components can influence through their expression across multiple cardiovascular cell types [11]. Robust experimental, clinical, and epidemiological evidence increasingly underscores the intricate link between vitamin D deficiency and the pathogenesis of cardiovascular disease [10, 3].

VITAMIN D DEFICIENCY AND CARDIOVASCULAR DISEASE RISK

For instance, vitamin D deficiency has been associated with cardiac inflammation, oxidative stress, alterations in cardiac structure, and systolic dysfunction, thereby contributing to the development and progression of various cardiovascular pathologies [3]. It is also associated with an increased incidence of atrial fibrillation, potentially due to its anti-inflammatory and anti-oxidative properties, and its negative regulation of the renin-angiotensin system [3]. Several studies have further demonstrated an inverse relationship between 25-hydroxyvitamin D levels and hypertension, suggesting that vitamin D may play a crucial role in blood pressure regulation through mechanisms such as inhibition of renin secretion [10]. This inhibitory effect on renin secretion can attenuate the renin-angiotensin-aldosterone system overactivation, a known contributor to hypertension and cardiac hypertrophy, as observed in vitamin D receptor-null mice [1, 12]. Furthermore, studies in rats have shown that vitamin D deficiency can lead to increased systolic blood pressure, enhanced superoxide anion production, and altered expression of cardiac genes involved in oxidative stress and myocardial hypertrophy [1].

PULSE WAVE VELOCITY AS A MEASURE OF ARTERIAL STIFFNESS

Arterial stiffness, quantified by PWV, serves as an independent predictor of cardiovascular morbidity and mortality, reflecting structural and functional changes in the arterial wall. This stiffening is often a consequence of vascular calcification, a highly regulated process involving osteochondrogenic differentiation of vascular smooth muscle cells, which significantly contributes to increased arterial stiffness and adverse cardiovascular outcomes [13].

DEFINITION AND MEASUREMENT OF PULSE WAVE VELOCITY

This measurement quantifies the speed at which the arterial pulse propagates along a segment of the arterial tree, with higher velocities indicating greater arterial stiffness. The gold standard for PWV measurement involves applanation tonometry to simultaneously record pressure waveforms at two arterial sites, typically the carotid and femoral arteries, allowing for the calculation of carotid-femoral PWV (cfPWV). The distance between these sites, divided by the time delay between the arrival of the pulse waves, yields the PWV, providing a robust indicator of large artery stiffness. Furthermore, elevated carotid-femoral PWV is consistently associated with an increased risk of future cardiovascular events, including myocardial infarction, stroke, and cardiovascular mortality, underscoring its prognostic significance in clinical practice. Beyond its prognostic value, PWV also serves as a sensitive biomarker for assessing the impact of various therapeutic interventions and lifestyle modifications on arterial health [12, 13, 14].

CLINICAL SIGNIFICANCE OF ARTERIAL STIFFNESS

Arterial stiffness, as indicated by increased PWV, is a well-established independent predictor of cardiovascular morbidity and mortality, reflecting the cumulative burden of structural and functional damage to the arterial wall. Pathologically increased aortic stiffness allows reflected waves from the periphery to return during cardiac systole, augmenting central systolic pressure and increasing the hemodynamic load on the left ventricle [8]. This increased load contributes to left ventricular hypertrophy and myocardial ischemia, thereby exacerbating cardiovascular risk [14]. The stiffening of large elastic arteries also accelerates the development of cardiovascular diseases and is exacerbated by conditions such as metabolic syndromes, diabetes mellitus, obesity, chronic kidney disease, atherosclerosis, and hypertension [15]. Moreover, heightened arterial stiffness is considered a summation of all key cardiovascular risk factors throughout an individual's life, serving as an independent marker for future cardiovascular events and mortality beyond traditional risk scores [16, 8]. Specifically, an increase of 1 m/s in PWV is associated with a 14% and 15% increased risk of cardiovascular events and mortality, respectively, highlighting the critical importance of mitigating arterial stiffness [17]. Carotid-femoral PWV is regarded as the most clinically relevant non-invasive measure of arterial health, especially as it reflects the compliance of the aortic arterial supply [18]. This makes cfPWV a key indicator for early detection of cardiovascular disease risk, enabling timely interventions to potentially decrease the incidence of overt disease [8]. It is widely recognized that arterial stiffness is a crucial component of vascular aging and a significant predictor of cardiovascular disease risks, making it an appropriate therapeutic target [19].

THE ASSOCIATION BETWEEN VITAMIN D AND PULSE WAVE VELOCITY

Considering the established role of arterial stiffness as an independent predictor of cardiovascular morbidity and mortality, the exploration of factors influencing PWV, such as vitamin D status, becomes critical for developing preventative strategies [20]. Research indicates that arterial stiffness, a multifactorial process involving extracellular matrix protein deposition and crosslinking, can be influenced by various factors, making vitamin D's potential role in modulating PWV a significant area of investigation [21].

Observational Studies: Numerous observational studies have explored the relationship between vitamin D levels and PWV, often revealing an inverse correlation where lower vitamin D concentrations are associated with greater arterial stiffness [7, 3]. Specifically, vitamin D deficiency has been linked to compromised vascular endothelial function, contributing to increased arterial stiffness and hypertension [1]. Furthermore, vitamin D deficiency is significantly associated with oxidative stress and inflammation, key contributors to arterial stiffness and chronic diseases associated with aging [22]. This association is particularly relevant given that vascular calcification, a highly prevalent condition in chronic kidney disease, directly contributes to arterial hardening, cardiac strain, and sudden cardiac death [10]. These observations highlight the potential for vitamin D supplementation to mitigate arterial stiffness, especially in populations at elevated cardiovascular risk. Moreover, a meta-analysis assessing the impact of vitamin D supplementation on flow-mediated dilation further supports its role in improving endothelial function, although direct evidence for PWV reduction remains less consistent [4]. Despite this, some studies have indicated that optimal vitamin D levels may exert a protective effect against arterial stiffening, potentially through direct regulation of the nuclear vitamin D receptor or indirect pathways involving the renin-angiotensin-aldosterone system [23].

Intervention Studies: Vitamin D Supplementation and PWV: While observational studies suggest a link, interventional trials investigating the direct impact of vitamin D supplementation on PWV have yielded mixed results, contributing to ongoing debate regarding its therapeutic efficacy in modulating arterial stiffness. This inconsistency underscores the complexity of vitamin D's systemic effects, which extend beyond specific vascular targets and may necessitate individualized approaches, potentially guided by pharmacogenomic insights, to ascertain clinical benefit [6]. For instance, some research indicates that vitamin D3 supplementation over a duration exceeding 12 weeks may lead to a significant reduction in arterial stiffness when compared to placebo or vitamin D2 [24]. Conversely, other studies have reported no significant changes in arterial stiffness markers, such as PWV, following vitamin D supplementation, particularly in cohorts with established cardiovascular disease or chronic kidney disease [4]. This divergence in findings may be attributable to variations in study populations, baseline vitamin D status, dosage regimens, and the duration of supplementation [25].

Potential Mechanisms Linking Vitamin D to Arterial Stiffness: Vitamin D is posited to influence arterial stiffness through various pathways, including the regulation of vascular smooth muscle cell function, inflammatory responses, and the renin-angiotensin-aldosterone system. Specifically, vitamin D can suppress renin gene expression, thereby mitigating the vasoconstrictive effects of the renin-angiotensin-aldosterone system and potentially improving arterial compliance [3, 26]. Activation of the vitamin D receptor has been shown to downregulate renin synthesis in juxtaglomerular cells and influence the expression of other components of the renin-angiotensin system within the kidney [27]. This mechanism suggests that vitamin D analogs could serve as renin inhibitors, akin to ACE inhibitors and ARBs, offering therapeutic potential for patients with hyperreninemia, metabolic syndrome, or hypertension [1]. Furthermore, vitamin D may also exert protective effects by modulating arterial wall calcification and reducing endothelial dysfunction, both critical determinants of arterial stiffness [28].

Methodological Considerations and Gaps in Current Research

A significant challenge in synthesizing research on vitamin D and PWV arises from the heterogeneity of study populations, encompassing diverse age ranges, health statuses, and comorbidities, which complicates direct comparisons and generalization of findings [6, 29]. Additionally, methodological differences in vitamin D assessment, PWV measurement techniques, and supplementation protocols further contribute to inconsistencies in the observed outcomes.

Study Design Heterogeneity: These variations include differing baseline vitamin D levels, ranging from severe deficiency to insufficiency, and a lack of standardized diagnostic criteria for defining vitamin D status across studies. Similarly, discrepancies in the duration and dosage of vitamin D supplementation protocols, as well as the specific form of vitamin D administered (e.g., D2 vs. D3), introduce further variability in study results [9].

Vitamin D Assay Variability: Moreover, the accuracy and precision of vitamin D assays themselves vary across laboratories, leading to potential misclassification of vitamin D status and influencing the interpretation of results. This variability underscores the critical need for harmonization of laboratory methods and the development of universal standards for vitamin D measurement to ensure data comparability and reproducibility. Furthermore, the choice between 25-hydroxyvitamin D [25D] and 1,25-dihydroxyvitamin D [1,252D] as the primary biomarker for assessing vitamin D status can significantly impact study outcomes, given their distinct biological roles and half-lives [6, 29, 30].

Confounding Factors: The interplay of parathyroid hormone, fibroblast growth factor 23, and Klotho also critically influences calcium and vitamin D metabolism, underscoring the necessity of considering these interactions when evaluating cardiovascular outcomes [3]. Furthermore, confounding factors such as genetic predispositions and lifestyle choices, including diet and physical activity, can significantly modulate the relationship between vitamin D and arterial stiffness, necessitating their careful consideration in study design and analysis. It is also crucial to acknowledge that various methods used to measure serum levels of vitamin D, such as mass spectrometry versus radioimmunoassay, contribute significantly to heterogeneity in study findings

[30].

FUTURE DIRECTIONS AND CLINICAL IMPLICATIONS

Given these complexities, future research should prioritize large-scale, well-designed randomized controlled trials that employ standardized methodologies for vitamin D assessment and PWV measurement to provide definitive evidence regarding the efficacy of vitamin D supplementation in cardiovascular health [31]. Such trials should also aim to stratify participants based on baseline vitamin D levels, genetic polymorphisms affecting vitamin D metabolism, and other relevant cardiovascular risk factors to identify specific subgroups that might benefit most from targeted interventions. Additionally, research into the microbiota's role in vitamin D metabolism and cardiovascular disease offers novel therapeutic avenues, particularly considering how altered intestinal flora due to vitamin D deficiency may impair B vitamin synthesis and contribute to a pro-inflammatory state [3]. Investigating the intricate interplay between vitamin D, the microbiome, and systemic inflammation could elucidate novel mechanisms underlying arterial stiffness and identify potential therapeutic targets.

Personalized Approaches to Vitamin D Supplementation*:* A deeper understanding of an individual's normal range for vitamin D, considering interactions with other cardiovascular risk mechanisms like parathyroid hormone, is crucial for defining precise impacts on cardiovascular risk and guiding supplementation strategies [32]. Moreover, accounting for genetic variants strongly associated with vitamin D status, deficiency, and metabolism as instrumental variables could mitigate biases inherent to traditional epidemiological studies, such as confounding and reverse causation, thereby strengthening causal inferences [29]. Longitudinal studies examining the dynamic changes in serum 25D levels over time are also essential to assess the sustained impact of vitamin D status on macrovascular complications and to determine if it can serve as a reliable biomarker for such conditions [33]. However, the inconsistencies in findings across various studies could be attributed to differing vitamin D preparations, variations in follow-up durations, patient compliance, and fundamental differences in study populations and sample sizes [1].

Longitudinal Studies and Causal Inference: Furthermore, while observational studies have illuminated associations between hypovitaminosis D and activated vasoconstrictive and hypertrophic mediators, thereby linking to endothelial dysfunction and elevated blood pressure, randomized controlled trials have yielded mixed results regarding vitamin D repletion's impact on cardiovascular function [34]. These discrepancies highlight the challenges in establishing causality solely through observational data and underscore the necessity of robust interventional studies to determine the utility of vitamin D therapy in cardiovascular disease management [10]. It is important to note that observational studies have consistently identified hypovitaminosis D as a risk factor for carotid atherosclerosis and other macrovascular complications, suggesting a role in cardiovascular pathology, potentially through mechanisms involving endothelial dysfunction, platelet aggregation, and inflammation [35, 29]. However, the precise mechanisms through which vitamin D exerts its cardiovascular protective effects, particularly regarding arterial stiffness, remain incompletely understood and warrant further investigation. Moreover, the potential for vitamin D receptor polymorphisms to modify the influence of serum 25D levels on macrovascular complications, particularly in prediabetic populations, presents a critical area for future genetic and functional research [33]. Therefore, further research with larger scale cohort studies and well-designed randomized controlled trials is essential to verify these relationships and unravel potential mechanisms [36].

CONCLUSION

This comprehensive review underscores the complexity of the relationship between vitamin D status and arterial stiffness, highlighting the need for rigorous research to translate observational findings into clinically actionable strategies. Future investigations should focus on large-scale, well-designed randomized controlled trials that incorporate standardized vitamin D assessment and advanced PWV methodologies to conclusively determine the efficacy of vitamin D supplementation in mitigating arterial stiffness.

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