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THE ROLE OF VITAMIN D IN PATHOGENESIS AND PREVENTION OF CARDIOVASCULAR DISEASES - A LITERATURE REVIEW

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ABSTRACT

Introduction: Cardiovascular diseases remain the leading cause of mortality worldwide, and its incidence is increasing with the population aging. A growing number of studies indicate that vitamin D deficiency, which is common in the global population, may play an important role in the pathogenesis of cardiovascular diseases by influencing inflammatory processes, oxidative stress, the renin-angiotensin-aldosterone system, calcium-phosphate metabolism, and endothelial function. As a result, vitamin D has become the subject of intensive analysis regarding its potential impact on the prognosis of patients with hypertension, heart failure, atherosclerosis, diabetes, and arrhythmias.

Aim of the study: The aim of the study was to present the current state of knowledge on the association between vitamin D deficiency and the risk and management of cardiovascular diseases, as well as to evaluate the role of vitamin D supplementation in the prevention and treatment of these diseases.

Material and methods: This paper provides a review of the literature including current experimental, observational, and randomized controlled studies on vitamin D metabolism, the significance of 25(OH)D deficiency, pathophysiological mechanisms, and the effects of supplementation. Included publications present the impact of vitamin D on endothelial function, oxidative stress, atherosclerosis, the neurohormonal system, glucose metabolism, and the risk of arrhythmia.

Results and Conclusions: A review of the literature indicates that vitamin D deficiency is associated with an increased risk of hypertension, heart failure, type 2 diabetes, atherosclerosis, and cardiac arrhythmias, including atrial fibrillation. The mechanisms of this effect include the action of VDR receptors in cardiomyocytes and vascular cells, modulation of the inflammatory response, reduction of oxidative stress, and regulation of the RAA system. Observational data suggest potential benefits of vitamin D supplementation, especially in high-risk groups, but large randomized clinical trials do not conclusively confirm that routine vitamin D administration reduces the number of major cardiovascular events in the general population. It is therefore necessary to individualize supplementation and conduct further well-designed studies to identify populations that may benefit most from therapy.

KEYWORDS

Vitamin D, Cardiovascular Diseases, Vitamin D Deficiency, Atherosclerosis, Supplementation, Cardiovascular Risk

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Introduction

Cardiovascular diseases, particularly coronary heart disease and stroke, are the leading cause of death worldwide. The incidence of cardiac events increases with age and is more common in men. It is estimated that as the population ages, the number of cardiovascular diseases will increase in developed countries [1,2]. Among the potential risk factors for cardiovascular disease, vitamin D deficiency is attracting increasing attention. In addition to the key role of vitamin D in maintaining calcium-phosphate homeostasis, deficiency of this compound has been linked to the development of numerous chronic diseases, including diabetes, hypertension, and chronic kidney disease [3]. Vitamin deficiency carries an increased risk of cardiovascular disease and mortality [4]. The action of vitamin D is largely mediated by the VDR receptor, for which $1,25(OH)_2D$ is the main ligand. As a ubiquitous transcription factor, VDR participates in numerous cellular processes, one of the most important of which is the control of calcium absorption in the small intestine [5]. Due to the presence of VDR in cardiomyocytes, vascular smooth muscle, and endothelium, it has become the subject of research aimed at elucidating the mechanisms by which vitamin D acts. Available studies in animal models have shown that high endogenous VDR expression protects the heart muscle from reperfusion injury and reduces its hypertrophy [6]. Vitamin D deficiency is associated with a poorer prognosis in the most common cardiovascular diseases—coronary artery disease, heart failure, and atrial fibrillation. Patients with these diagnoses are recommended to take vitamin D supplements, even though current studies on the effectiveness of this treatment for vascular diseases have conflicting results [3].

This review analyzes the current state of knowledge on vitamin D deficiency and its potential impact on the prognosis and course of cardiovascular diseases. Particular attention was paid to the pathophysiological mechanisms linking vitamin D deficiency to the development of endothelial dysfunction, hypertension, atherosclerosis, and heart failure. The available clinical data on the impact of vitamin D supplementation on the risk of developing and progressing cardiovascular diseases were also discussed. The aim of this paper is to provide a synthetic overview of the current scientific evidence and to assess the potential role of vitamin D supplementation in the prevention and treatment of cardiovascular diseases.

Metabolism of vitamin D

Vitamin D is a secosteroid derived from animal cholesterol (cholecalciferol, vitamin D3) and plant ergosterol (ergocalciferol, vitamin D2) [7]. Endogenous vitamin D is produced in a two-step non-enzymatic reaction in the keratinocytes of the epidermal plasma membrane under the influence of exposure to ultraviolet B radiation. The first step involves the conversion of 7-dehydrocholesterol into a thermodynamically unstable 9,10-secosteroid molecule (precholecalciferol). The second stage, which takes place at body temperature (approximately 37°C), involves thermal isomerization, which leads to the formation of a thermally stable molecule, cholecalciferol. Sunlight is the main source of its acquisition [1,8]. Smaller amounts can be found in food, mainly in fatty fish, mushrooms, egg yolks, beef kidney, pork liver, or cod-liver oil [1]. Hydroxylation occurs in the liver, resulting in the formation of 25-hydroxyvitamin D [25(OH)D], which is then converted to calcitriol 1,25 (OH)2D in the proximal convoluted tubules of the kidneys thanks to the activity of the enzyme 1-α-hydroxylase [2]. To analyze vitamin D concentration in the blood, it is useful to measure 25(OH)D, which has a long half-life that ranges from 2 to 3 weeks. This conversion is also possible in the cardiovascular system with the participation of endothelial cells, macrophages, and smooth muscle cells. In the heart, vitamin D-binding receptors (VDR) are found in ventricular cardiomyocytes and fibroblasts [7,9]. Such active vitamin D has autocrine and paracrine effects [2]. It is essential for maintaining normal vascular tone and has anti-fibrotic and anti-hypertrophic properties. Regulates calcium flow, thereby increasing myocardial contractility [7,10]. Reduces inflammation, inhibits the proliferation of vascular smooth muscle, and regulates the renin-angiotensin-aldosterone system. It has the ability to release anti-inflammatory cytokines and contributes to the reduction of inflammatory mediators [2]. Calcitriol is released into the bloodstream, where it binds to protein. It has lipophilic properties, allowing it to penetrate the cell membrane and bind to receptors located in the cytoplasm or nucleus of target cells [7].

Vitamin D deficiency

Vitamin D deficiency may increase the risk of developing chronic debilitating diseases such as type 2 diabetes, metabolic syndrome, hypercholesterolemia, chronic kidney disease, gastrointestinal malabsorption syndrome, parathyroid disorders, obesity, and hypertension. It has been reported that 65% of older people worldwide do not reach the minimum laboratory value of 25-hydroxyvitamin D [25(OH)D] of 20 ng/ml. This is the most common nutritional deficiency, affecting approximately one billion people worldwide. Newborns, adolescents, pregnant women, and breastfeeding women are also at risk. Deficiency is also observed in young people [2,11]. Many factors can contribute to its reduction, such as nutrition, physical activity, exposure to sunlight, skin color, reduced absorption, and acquired and hereditary metabolic disorders [1,5]. In addition, vitamin D deficiency can cause inflammation in the fatty tissue surrounding the heart and in the walls of blood vessels through direct interaction with nuclear factor kappa beta (NFκB), which intensifies inflammatory reactions and promotes the development of atherosclerosis and cardiovascular diseases [1]. This is associated with increased arterial stiffness, left ventricular hypertrophy, cerebrovascular events, hypertension, and endothelial dysfunction—both in patients with chronic kidney disease and in healthy individuals. This may indicate that vitamin D plays a protective role in the cardiovascular system, helping to limit changes leading to heart failure [7,12].

Screening tests and vitamin D level tests

Screening tests are not clinically useful and are very expensive. They have been used mainly in patients suffering from musculoskeletal disorders. Vitamin D concentrations can be measured using immunoassays (chemiluminescent or immunoenzymatic) or the more accurate method of liquid chromatography coupled with mass spectrometry (LC-MS or LC-MS/MS), which provides more reliable results. The report should indicate the reference material used and the unit of measurement – preferably in moles per liter (mol/l), or possibly in parallel in ng/ml. The laboratory should also establish its own reference ranges appropriate for the method used. In addition, the so-called critical difference must be taken into account, i.e., the minimum change in concentration between two consecutive tests of the same patient, which indicates an actual change in level and not just natural fluctuations or measurement errors [5].

Vitamin D and heart failure

The number of patients suffering from heart failure is constantly growing due to the aging population, becoming a global problem [13]. The pathogenesis of heart failure involves many interdependent processes, including hemodynamic disorders, activation of neurohormonal systems, intensification of the inflammatory response, and changes in the availability of key micronutrients. These factors may explain the limited effectiveness of current therapies in improving clinical outcomes [3]. The most recent observational data indicate that low levels of 25-hydroxyvitamin D may be of significant prognostic importance in patients with heart failure [13]. In one clinical trial, patients with heart failure and reduced ejection fraction were divided into groups receiving vitamin D or placebo for one year. Although no differences in physical performance were reported, those supplementing with vitamin D showed improvements in heart function, including an increase in ejection fraction and favorable changes in left ventricular structure [10]. In another study involving patients with the same type of heart failure, short-term vitamin D supplementation reduced renin-angiotensin system activity, which may indicate its protective effect in the context of cardiomyopathy [10]. A meta-analysis by Wang et al. involving nearly 6,000 patients showed that vitamin D deficiency is associated with a significantly increased risk of overall mortality, suggesting that vitamin D status may reflect disease severity and overall metabolic and inflammatory burden in this population. Although the analyzed data did not confirm a clear association between 25-(OH)D concentration and the risk of rehospitalization, the authors emphasized the high heterogeneity of research projects and the limitations resulting from the observational nature of the available analyses. These results support the hypothesis that vitamin D deficiency may serve as an independent prognostic marker in heart failure, while also pointing to the need for well-designed randomized trials to assess whether supplementation can improve survival and slow disease progression [13].

Vitamin D and diabetes mellitus

There is a steady increase in the incidence of diabetes. Studies show that in people with diabetes, the active form of vitamin D – 1,25$(OH)_2$D – limits the absorption of cholesterol by macrophages in endothelial cells and reduces its excessive accumulation, affecting the activity of the endoplasmic reticulum [2]. Maintaining adequate vitamin D levels in infants, especially during the first six months of life, is associated with a lower risk of developing type 1 diabetes. On the other hand, symptoms suggestive of rickets may increase this risk. It has been noted that giving children cod liver oil, which is rich in vitamin D, in early life reduces the likelihood of developing diabetes [14]. Pancreatic beta cells contain vitamin D receptors (VDR) that stimulate insulin secretion [1]. Additionally, inhibition of its activity promotes the transformation of M2 macrophages into M1 macrophages, suggesting that vitamin D may modulate their function and represent a potential direction for anti-atherosclerotic therapy. Macrophages that enter the subendothelial space respond to local signals and differentiate into different cell types. Within the atherosclerotic plaque, M1 macrophages have receptors that enable the migration of immune cells from the lesion area, while M2 macrophages – activated, among others, by interleukins IL-4 and IL-10 – alleviate inflammatory reactions, support collagen synthesis and contribute to fibrosis processes [2].

Vitamin D and hypertension

In recent years, attempts have been made to understand the impact of the vitamin D receptor on the cardiovascular system. It turns out that its absence leads to increased blood pressure as a result of increased secretion of renin and angiotensin II [15]. Studies evaluating the impact of VDR ablation on the circulatory system have shown that it leads to an increase in the development of atherosclerosis, hypertension and myocardial hypertrophy, probably due to local activation of macrophages and stimulation of the renin-angiotensin-aldosterone system (RAAS) [2,15]. The excretion of angiotensinogen in urine is considered an indicator of intrarenal RAAS activity, which is significantly associated with renal dysfunction and the occurrence of hypertension. Patients with renal failure often have low vitamin D levels. It has been established that patients with heart failure and impaired renal function had significantly lower 25(OH)D concentrations than individuals with normal filtration. In addition, increased oxidative stress was observed. An analysis taking into account age, gender and BMI confirmed that vitamin D deficiency may increase oxidative stress and indirectly increase RAA activation, despite the use of drugs that block this system. It has been shown that vitamin D supplementation can reduce oxidative stress and its effects. Therefore, combining RAA-blocking drugs with vitamin D or other antioxidants may be beneficial in patients with heart and kidney failure. Vitamin D may also contribute to slowing the deterioration of filtration in people with chronic kidney disease using RAA blockade [16].

Atherosclerosis and oxidative stress

The main factor contributing to the development of atherosclerosis is the overproduction of reactive oxygen species (ROS), which lead to oxidative stress. These are highly reactive oxygen-containing particles that are formed in the oxygen reduction reaction. This group includes free radicals such as superoxide (O2-), hydroxyl species (HO) and non-free radicals such as hydrogen peroxide (H2O2). RFTs play an important role in regulating vascular function. They are essential for maintaining normal signalling pathways that are involved in controlling cellular processes such as proliferation, inflammation, differentiation and apoptosis. However, their excess promotes processes that lead to atherosclerosis, such as inflammation, endothelial damage and lipid metabolism disorders. The main enzymes responsible for the production of reactive oxygen species include NADPH oxidases, xanthine oxidase, nitric oxide synthases and mitochondria. Their action is usually balanced by antioxidant systems, but in vascular diseases this balance may be disturbed, leading to increased oxidative stress [17].

Vitamin D and atherosclerosis

Atherosclerosis is a chronic disease where the deposition of fibrofatty lesions in the artery wall leads to the formation of atherosclerotic plaques [18]. Their presence is associated with chronic tissue ischaemia [18,19]. The rupture of an atherosclerotic plaque may be accompanied by the formation of a thrombus, which, by blocking blood flow through the arteries, leads to acute ischaemia and tissue necrosis. This mechanism is observed in acute ischaemic stroke or acute coronary syndrome [19]. Data from observational and interventional studies indicate that vitamin D deficiency is associated with an adverse lipid profile, while its normal concentration correlates with more favourable lipid parameters [19]. Patients with insufficient vitamin

D levels are more likely to suffer from hypertension, dyslipidaemia, insulin resistance and obesity [20]. A study by Carbone et al. (2023) provides an in-depth insight into the role of vitamin D in the pathogenesis of atherosclerosis, indicating that its action goes beyond the classic effects on calcium and phosphate metabolism. A local vitamin D system is present within atherosclerotic plaques, which acts through intracrine, autocrine and paracrine mechanisms, regulating the cellular functions of macrophages, vascular smooth muscle cells and dendritic cells [8]. In case of insufficient concentration of this vitamin, there is an increase in the number of vascular cell adhesion molecules and E-selectin [19]. Vitamin D inhibits the formation of foam cells by activating the VDR receptor, including by reducing endoplasmic reticulum stress and the expression of lipoprotein receptors, and supports autophagy via the PTPN6/SHP-1 pathway [8].

Vitamin D and arrhythmias

A growing number of studies indicate that low vitamin D levels are associated with an increased risk of atrial fibrillation (AF) and postoperative arrhythmias, prompting consideration of the role of vitamin D as a potential protective factor. A meta-analysis of 13 studies (74,885 participants) found that vitamin D deficiency (< 20 ng/ml) was associated with an increased risk of AF (RR = 1.23; 95% CI: 1.05–1.43), and each 10 ng/ml increase in concentration was associated with a 12% reduction in risk (RR = 0.88) [21]. Similarly, a more recent prospective meta-analysis from 2023 showed that vitamin D deficiency was associated with a ~12% higher risk of AF, and a 10 ng/ml increase in concentration was correlated with a reduced risk (HR = 0.95) [22]. On the other hand, interventional data are still limited and show mixed results: for example, an earlier meta-analysis of RCTs did not show a significant effect of vitamin D supplementation on the prevention of 'new' AF in the general population [23]. Interestingly, in the context of arrhythmias after cardiac surgery, vitamin D supplementation before surgery appears to reduce the incidence of POAF: a recent analysis of four RCTs (694 patients after CABG) found that vitamin D administration reduced the risk of POAF by ~45% (RR = 0.55; 95% CI 0.40–0.76) [24]. This suggests that the benefit may be evident in high-risk groups (e.g., patients after heart surgery), although further well-designed studies with large numbers of participants and longer follow-up periods are needed to confirm whether vitamin D can indeed be an effective component of arrhythmia prevention strategies.

Supplementation

Vitamin D supplementation is sometimes seen as a potential strategy for influencing the risk of cardiovascular disease, but the results of studies to date remain inconclusive. On the one hand, observational and review data indicate that low 25(OH)D concentrations correlate with an increased incidence of cardiovascular disease, suggesting a possible role for vitamin D in its pathogenesis through modulation of inflammation, influence on glucose metabolism or regulation of blood pressure [25,26]. On the other hand, the results of randomised clinical trials do not confirm that routine vitamin D supplementation significantly reduces the risk of major cardiovascular events. For example, in a large D-Health Trial involving more than 21,000 people, only a small, statistically uncertain trend of a decrease in the risk of cardiovascular events (HR 0.91) was observed, as the confidence interval included the possibility of no effect [27]. Similarly, meta-analyses of intervention studies have not shown a significant reduction in the risk of MACE or cardiovascular mortality [28]. Consequently, although vitamin D deficiency is common and associated with a poorer prognosis, current evidence does not justify its routine use solely for the prevention of cardiovascular disease.

The term 'dose' of vitamin D refers to the amount of this vitamin contained in one tablet or capsule — most often in the form of cholecalciferol, although other forms are also available, such as ergocalciferol, calcifediol and eldecalcitol. This amount is given in micrograms (µg) or international units (IU), with 10 µg corresponding to 400 IU. Oral administration of vitamin D is the most beneficial route of administration. Cholecalciferol is the most commonly chosen form. However, in some situations, parenteral administration of vitamin D analogues, i.e. calcitriol and calcifediol, is necessary. In this case, cholecalciferol proves to be the safest and least demanding to monitor. This form is administered to people with impaired absorption in the gastrointestinal tract, i.e. in inflammatory bowel diseases, pancreatic insufficiency, short bowel syndrome, coeliac disease, after bariatric surgery, and in situations requiring total parenteral nutrition. Calcitriol is used when the active metabolite is not easily produced in vivo [5,29]. The dose of cholecalciferol is clinically significant because it affects the concentration of 25(OH)D in the blood, which is the main indicator of vitamin D status and is associated with various health effects. In practice, there are loading doses – used to quickly raise low vitamin D levels – and maintenance doses, which are intended to maintain the achieved concentration. However, the efficacy and safety of large, infrequent doses are questionable, as some studies indicate the

possibility of adverse effects. For this reason, daily dosing is preferred, as it provides more stable and safer results.

The body's response to a given dose of vitamin D may vary depending on individual factors such as body weight, absorption, diet, obesity, and the activity of enzymes and transport proteins. To determine the need for vitamin D supplementation, the total intake of vitamin D from various sources, i.e. natural foods, fortified foods and skin synthesis, should be taken into account [30]. According to the National Academy of Medicine, the recommended daily intake of vitamin D is between 400 and 800 IU, with an upper safe limit of 4000 IU. However, some experts suggest that the actual safety limit may be slightly lower. Doses of 400–800 IU per day are usually sufficient to prevent deficiencies and maintain normal calcium metabolism in healthy individuals. Higher doses may be associated with a risk of toxicity, although some studies have used up to 10,000 IU per day without observing any adverse effects [5]. It has been established that vitamin D3 supplementation at a monthly dose of 60,000 IU for five years significantly reduces the incidence of cardiovascular events, with particular emphasis on myocardial infarction and coronary revascularisation [27]. In a large population study of people without previous cardiovascular disease, the risk of developing heart failure was lower among participants receiving vitamin D compared to the control group. However, a meta-analysis of multiple studies did not clearly confirm this effect, probably due to large differences in the designs and populations of individual studies [10]. Vitamin D plays a very important role in maintaining the body's homeostasis. Maintaining normal levels has a beneficial effect on metabolic processes, including the cardiovascular system, ensuring its protection. Prophylactic supplementation and treatment of cardiovascular diseases with vitamin D does not prevent their occurrence [8,31]. Reasonable and individually selected dosages allow for maximum health benefits while maintaining the safety of the therapy [5].

Conclusions

Vitamin D deficiency is a common health problem with broad clinical significance that can affect the development and progression of cardiovascular disease. An analysis of available studies indicates that low 25(OH)D levels are associated with an increased risk of hypertension, heart failure, atherosclerosis, diabetes and cardiac arrhythmias, including atrial fibrillation and post-operative arrhythmias. Mechanistic data suggest that vitamin D acts through VDR receptors in the heart and blood vessels, modulating inflammatory processes, oxidative stress, endothelial function, the renin-angiotensin-aldosterone system, and calcium and lipid metabolism. Vitamin D supplementation in observational and some interventional studies shows potential benefits, especially in high-risk groups, but large randomised clinical trials do not provide clear evidence that routine vitamin D administration reduces the risk of cardiovascular events in the general population. The conclusion from the analysed literature is that an individual approach to supplementation is necessary, taking into account vitamin D levels, the presence of comorbidities and potential drug interactions. Further well-designed clinical trials are needed to precisely determine which populations may benefit from vitamin D supplementation in the prevention and treatment of cardiovascular disease.

Disclosure

Author's contribution

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