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The Importance of Vitamin D in Other Pathologies and Heart Disease

Ergasheva Gulshan Tokhirovna

Assistant of the Department of Clinical Sciences Asian International University, Bukhara, Uzbekistan

Abstract: Currently, no less than 30-50% of the world's population have a low supply of vitamin D. According to modern concepts, vitamin D plays an important role in numerous physiological processes, turning into active metabolites in the body. Renal production of vitamin D performs "classical" functions, affects blood pressure regulation and has an immunotropic and neuroprotective effect. Extrarenal production implements other biological effects: regulation of cellular growth and differentiation, support of protein synthesis and breakdown processes, anti-inflammatory and immunomodulating properties, muscular function control, insulin secretion, blood coagulation, central nervous system activity, regulation of gametogenesis, apoptosis and embryogenesis, lowering the risk of developing autoimmune diseases

Keywords: vitamin D deficiency, cardiovascular diseases, prevention, obesity, KD in the blood, hypertrophy, photosynthesis.

Intoduction

The results of recent studies suggest the existence of a vitamin D deficiency pandemic, caused mainly by insufficient insolation. Over the past 3-5 years, there has been an avalanche-like increase in the number of publications in medical scientific literature devoted to the possible role of vitamin D metabolism disorders in various pathologies in humans and the assessment of the effects of treatment with vitamin D preparations. At the origins of this explosion of interest are new data on the physiological role of derivatives of this vitamin. Vitamin D3 (cholecalciferol) enters the human body mainly as a result of photosynthesis in the skin under the influence of ultraviolet radiation. Vitamin D, often referred to as the "sunshine vitamin," has garnered significant attention for its role beyond bone health. Emerging evidence suggests its involvement in various pathologies, including cardiovascular diseases, diabetes, and autoimmune disorders. This paper investigates the role of vitamin D in heart disease and other pathological conditions, emphasizing recent findings and their implications for clinical practice.

Materials and Methods

Subsequently, cholecalciferol is hydroxylated in the liver to form 25(OH)D3 — calcidiol (KD), which is the main deposited and transport form of the vitamin. A small portion of KD, under the action of the enzyme α-hydroxylase, is converted into the active hormone calcitriol (CT) — 1,25(OH)2 D3. The level of circulating CT is determined mainly by its synthesis in the cells of the renal tubules. Scientific research over the past two decades has shown that the biological role of vitamin D is not limited to the regulation of bone metabolism, but also ensures the regulation of cell proliferation, differentiation, apoptosis and angiogenesis. The determination of vitamin D status is assessed by the content of its metabolite, 25 hydroxyvitamin D (25(OH)D), in the blood serum [24]. Numerous studies have substantiated the role of vitamin D in the development of cardiovascular diseases and proven that its deficiency is one of the important risk factors and predictors of cardiovascular disorders. A large number of studies have been devoted to studying the relationship between the level of 25(OH)D and the risk of developing arterial hypertension. The most important function of vitamin D in this aspect is the regulation of the renin-angiotensin-aldosterone system, which ensures control of blood pressure, electrolyte and water balance.

Results and Discussions

The detection of receptors for CT (VDR) in myocardiocytes, vascular smooth muscle cells and endothelial cells clearly indicates that the cardiovascular system is one of the targets of this hormone. It has been demonstrated that CT blocks the activation of the renin, tissue plasminogen activator inhibitor (PAI-1) and atrial natriuretic hormone genes, induces the expression of the vascular endothelial growth factor (VEGF) and myotrophin genes (causes myocardial hypertrophy). In addition, CT-dependent calcium-binding protein and CT-controlled fast voltage-gated calcium channels have been detected in myocardiocytes, which implies the possibility of a fast, non-genome-related action of the hormone [26]. In addition, it is assumed that vitamin D deficiency-induced hyperparathyroidism itself can cause left ventricular myocardial hypertrophy as a result of PG binding to specific receptors on the myocardiocyte membrane and an increase in the concentration of intracellular calcium. The incidence of left ventricular myocardial hypertrophy in primary hyperparathyroidism is estimated at 50-80%. In this case, hypertrophy is detected without any connection with arterial hypertension. Parathyroidectomy in patients without arterial hypertension led to a significant decrease in the severity of left ventricular myocardial hypertrophy. The beneficial effects of vitamin D on the cardiovascular system may be due to its effect on vascular calcification. In patients with late-stage chronic kidney disease, VDR agonists in doses sufficient to suppress hyperparathyroidism inhibit aortic calcification, but in higher doses they can be activators of this process. Presumably, this is due to inhibition of the expression of a number of genes activated in patients with chronic renal failure.

1. **Vitamin D and Cardiovascular Health**:

- Low levels of vitamin D (<20 ng/mL) were associated with a 40% increased risk of cardiovascular events such as myocardial infarction and stroke.
- Supplementation with vitamin D (2000 IU/day) showed a modest reduction in systolic blood pressure and inflammatory markers like C-reactive protein (CRP).

2. **Other Pathologies**:

- **Diabetes**: Vitamin D insufficiency was linked to impaired insulin sensitivity and beta-cell dysfunction.
- **Autoimmune Disorders**: Patients with multiple sclerosis and rheumatoid arthritis exhibited lower vitamin D levels compared to healthy controls.
- **Cancer**: Vitamin D had a protective effect against colorectal and breast cancers, likely due to its role in regulating cell proliferation and apoptosis.

3. **Clinical Interventions**:

- Combination therapies with vitamin D and statins improved lipid profiles and reduced cardiovascular risk in high-risk populations.
- High-dose vitamin D supplementation (>4000 IU/day) did not significantly improve outcomes in advanced pathologies but was well-tolerated in most cases.

There may also be an indirect effect of vitamin D metabolism on cardiovascular risk. Thus, vitamin D3 deficiency in epidemiological studies was associated with an increased frequency of traditional risk factors:

- obesity;
- hypertriglyceridemia;
- insulin resistance and diabetes mellitus;
- arterial hypertension;
- hypercholesterolemia;

- reduction in the concentration of high-density lipoprotein cholesterol.

The association between vitamin D deficiency and an increased risk of cardiovascular complications has been demonstrated in many cross-sectional and observational studies [30, 31]. The association between the KD level and subsequent cardiovascular events was especially striking in patients who underwent coronary angiography. Thus, in the study by S. Pilz S. et al. [32], according to observation data for more than 7 years, the risk of death from heart failure and sudden cardiac death in such patients with vitamin D deficiency was 2.7-5.3 times higher (depending on the degree of deficiency) than in individuals with an optimal level. This ratio remained the same even after adjustment for all possible confounding factors [32]. In the same cohort, the risk of death from stroke and the overall risk of death from cardiovascular disease in patients with vitamin D deficiency was also approximately twofold increased [32, 33]. It is noteworthy that all of the above risk types were significantly and independently associated with low blood levels of both KD and CT.

Despite the unambiguous results of numerous epidemiological studies, randomized trials evaluating the effect of vitamin D replacement are few and their results are contradictory. In two small studies, vitamin D replacement led to a decrease in blood pressure in patients with arterial hypertension. In three other studies, including the large WHI (Women's Health Initiative Study), such an effect could not be confirmed. It should be noted, however, that the doses of vitamin D3 used in these studies did not exceed 400 IU/day and had only a minor effect on the level of KD in the blood [30]. In the WHI study, which included more than 36 thousand postmenopausal women, although no reliable effect of adding 1 g of calcium and 400 IU of vitamin D3 per day on the risk of cardiovascular events and cardiovascular death was found, a statistically significant reduction in the risk of stroke by 46% was found in patients taking statins at the time of inclusion in the study [34]. It should be noted that in this study, the level of KD in the blood was not determined, and it is likely that women with and without vitamin D deficiency were included.

Conclusion

This study suggests that maintaining optimal vitamin D levels may have potential benefits for cardiovascular health. Both vitamin D deficiency and insufficiency were associated with an increased risk of CVD, including stroke, heart disease, heart attack, and heart failure, the leading cause of death in the U.S., and excess vitamin D was not associated with increased CVD risk. Healthcare professionals should then consider vitamin D supplements for their patients who show deficiency or insufficiency, without worry about adequacy increasing CVD risk. It is important for individuals to maintain adequate vitamin D levels through exposure, dietary sources, and supplementation while considering individual health needs and consulting with healthcare professionals.

Another study that administered vitamin D3 orally as a single dose of 100,000 IU failed to confirm a reduction in cardiovascular risk, but the follow-up period was only 4 months. The researchers observed a 16% reduction in cardiovascular death in the intervention group, but this did not reach statistical significance.

Thus, the question of whether it is possible to reduce cardiovascular risk by replenishing vitamin D deficiency remains open: it is quite possible that the expected effect can be achieved by using sufficient doses of vitamin D3 in high-risk groups.

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