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SCIENCE IN THE MODERN WORLD: INNOVATIONS AND CHALLENGES

PROCEEDINGS OF XIII INTERNATIONAL SCIENTIFIC AND PRACTICAL CONFERENCE SEPTEMBER 4-6, 2025

TORONTO 2025

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THE PROBLEM OF CHOLECALCIFEROL DEFICIENCY AMONG PATIENTS WITH ARTERIAL HYPERTENSION

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Annotation: arterial hypertension (AH) remains the leading modifiable risk factor of cardiovascular diseases, stroke, and chronic kidney disease. Its high prevalence and close connection with cardiovascular diseases and premature death determine the relevance of this problem for health care systems worldwide [1, p. 4; 2, p. 5].

Recently, vitamin D deficiency has acquired the status of a pandemic, affecting more than a billion people regardless of age and ethnic belonging. This has generated a central scientific hypothesis: whether this connection is causal, or the observed association is only an epiphenomenon reflecting other risk factors, such as obesity, sedentary lifestyle, and general health condition? During the recent time, there is a growing body of data indicating a significant connection between low vitamin D levels and various cardiovascular diseases and conditions. [3, p. 5; 4, p. 5].

Keywords: vitamin D, endothelium, hypertension, cardiovascular, renin.

Aim. The purpose of the work is to analyze scientific sources on the problem of cholecalciferol deficiency in patients with arterial hypertension.

Materials and Methods. We analyzed the scientometric databases PubMed, Scopus, Web of Science and conducted a retrospective analysis of literature sources. The most relevant sources on this topic were selected for analysis.

Results and Discussion. Traditionally, vitamin D was associated with the regulation of calcium-phosphorus metabolism and the health of bone tissue. However, modern endocrinology considers it as a steroid hormone with a wide

spectrum of pleiotropic effects, which are realized through the presence of specific vitamin D receptors in most body tissues, including the cells of the cardiovascular and immune systems [5, p. 5].

Vitamin D exists in two main forms: ergocalciferol (vitamin D_2), which is synthesized in plants and fungi, and cholecalciferol (vitamin D_3), which is produced in the skin of humans and animals under the influence of ultraviolet radiation, and is also obtained from animal-derived foods. Cholecalciferol is the biologically more active form, approximately twice as potent as ergocalciferol [6, p. 5].

The assessment of the body's vitamin D supply is carried out by measuring the concentration of 25-hydroxyvitamin D (25(OH)D) in blood serum. There are consensuses of international and Ukrainian expert societies regarding the classification of vitamin D status, which is fundamental for the interpretation of research results and clinical practice. Deficiency is defined as a level <20 ng/ml (<50 nmol/L), and insufficiency as a level of 20–29 ng/ml (50–74 nmol/L) [7, p. 5; 8, p. 5]

The causes of vitamin D deficiency are multifactorial and include: insufficient insolation due to living in high latitudes, seasonality (autumn–winter), limited sun exposure, wearing closed clothing, and the use of sunscreens; insufficient dietary intake with a vegetarian diet or unbalanced nutrition; impaired metabolism in liver and kidney diseases that reduce the activity of 25-hydroxylase and 1α-hydroxylase; increased catabolism due to the use of certain medications (for example, glucocorticoids or statins); and individual factors such as age and obesity [9, p. 6; 10, p. 6; 11, p. 6]

The biological plausibility of the connection between low vitamin D levels and elevated blood pressure is supported by several powerful pathophysiological mechanisms. These mechanisms are not mutually exclusive and are likely to act synergistically, creating conditions for the progression of arterial hypertension [12, p. 6].

To date, the most well-studied mechanism is the role of vitamin D as a negative endocrine regulator of the renin–angiotensin–aldosterone system, the key

system for the regulation of blood pressure and water–electrolyte balance. Observational studies consistently reveal an inverse correlation between 25(OH)D levels and plasma renin activity. This indicates that in vitamin D deficiency its inhibitory effect on renin synthesis disappears, leading to chronic hyperactivation of the RAAS, increased production of angiotensin II and aldosterone, which, in turn, causes vasoconstriction, sodium and water retention, and, as a consequence, elevated blood pressure [13, p. 6; 14, p. 6]

A healthy endothelium - the inner lining of blood vessels - plays a key role in regulating vascular tone, primarily through the production of a powerful vasodilator, nitric oxide (NO). Endothelial dysfunction, characterized by reduced bioavailability of NO, is an early event in the pathogenesis of arterial hypertension. Vitamin D deficiency contributes to the development of endothelial dysfunction. Oxidative stress, that is, an excess of reactive oxygen species, leads to the destruction of NO and the suppression of eNOS activity. Vitamin D has antioxidant properties; in particular, it enhances the activity of antioxidant enzymes such as superoxide dismutase. In its deficiency, this protective mechanism is weakened [15, p. 6; 16, p. 6].

Currently, arterial hypertension is increasingly considered a state of chronic low-grade inflammation. Vitamin D is a powerful immunomodulator with pronounced anti-inflammatory properties. It stimulates the production of anti-inflammatory cytokines, in particular interleukin-10. Vitamin D also inhibits the activation of the key pro-inflammatory transcription factor NF-κB, which leads to a reduction in the production of the pro-inflammatory cytokine interleukin-6, which directly contributes to the development of endothelial dysfunction. In the study by K.L. Jablonski et al., it was shown that in patients with vitamin D deficiency there is increased expression of NF-κB and IL-6 in endothelial cells, which correlates with reduced endothelium-dependent vasodilation [17, p. 7; 18, p. 7].

Conclusions. Thus, currently, certain interrelations between cholecalciferol deficiency and the pathophysiological mechanisms of arterial hypertension have been established. There is a strong biological basis, supported by experimental data, that

explains how vitamin D deficiency can affect blood pressure regulation through its influence on the renin–angiotensin–aldosterone system, endothelial function, and inflammatory processes. These mechanisms are reflected in the associations observed in numerous studies.

Future Research Directions. It is possible that correction of vitamin D deficiency may provide a clinically significant antihypertensive effect in those who have both established arterial hypertension and pronounced hypovitaminosis D. For clinical practice, this means that although vitamin D is not an antihypertensive drug, ensuring its sufficient level in patients with hypertension is a safe and inexpensive intervention that has the potential to improve treatment outcomes.

Conflicts of interest: Authors have no conflict of interest to declare.

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