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THE CONNECTION BETWEEN LUPUS AND VITAMIN D

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Abstract

The review presents literature data on the frequency of hypovitaminosis D in systemic lupus erythematosus (SLE), analyzes the relationship of clinical and laboratory indicators of the disease with the level of D level, and considers the possibilities of therapeutic use of its metabolites. The antiresorptive, anti-inflammatory and immunomodulatory effects of vitamin D metabolites justify the priority of their use in chronic inflammatory diseases in combination with long-term basic methods.

Key words

Vitamin D; systemic lupus erythematosus; hypovitaminosis D.

INTRODUCTION

In the last decade, there has been increased interest in the potential role of vitamin D in autoimmune diseases and the possibility of its therapeutic use. The results of numerous studies have formed a firm belief that vitamin D is not just a vitamin, but a steroid hormone that plays a huge role in human biology and is no less important for bone metabolism than calcium [1].

Vitamin D exists in two forms: vitamin D3 (cholecalciferol) and vitamin D2 (cholecalciferol). ergocalciferol). Sources of vitamin D for the body are food and endogenous synthesis. Vitamin D2 is produced by some types of mushrooms under the influence of ultraviolet light. ergosterol [2]. Humans synthesize vitamin D3, its most natural form. The main part of vitamin D (more than 80%) is formed in the body by synthesis in the skin under the influence of sunlight - ultraviolet radiation (UVR). Ultraviolet waves with a length of 290-315 nm (B) upon contact with the skin convert 7-dehydrocholesterol by breaking one of the cyclopentane-perhydrophenanthrene rings into provitamin D3, which is converted into vitamin D3. Only 20% of the vitamin D the body needs comes from food [3]. Vitamin D, synthesized in the skin or supplied with food and medications, is metabolized in the liver to 25(OH)-vitamin D (calcidiol), the content of which in the blood serum determines the patient's vitamin D status [4, 5]. 25(OH)D, complexed with vitamin D binding protein, is then transported to the kidneys, where the final phase of activation of vitamin D into 1,25-dihydroxyvitamin B (calcitriol) occurs. 1,25(OH)2B interacts with target organs in body tissues by binding to the nuclear vitamin D receptor (VDR), which belongs to the superfamily of ligand-activated transcription factors [6, 7]

Table 1. Serum 25(OH)D levels and associated conditions

Status	Nmol/l ng/ml	Nmol/l ng/ml
Deficiency (rickets, osteomalacia).	<30	<12
Refusal	30-50	12-20
Norm	>50	>20
Excess _	>125	>50
D intoxication	>374	>150

The main function of vitamin D is the interaction between the processes of bone tissue resorption/formation and calcium homeostasis [8]. In recent years, additional effects of vitamin D have been discovered that are not related to the regulation of calcium homeostasis, primarily the

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effect on cell differentiation and proliferation. It has been established that 1-hydroxylation of 25(OH)D occurs not only in the kidneys, but also in other tissues, while the extrarenally formed 1,25(OH)D2 functions autocrine. Extrarenal 1ahydroxylase activity is influenced by cytokines and growth factors that optimize 1,25(OH)D2 levels for their cell-specific action. Extrarenal 1,25(OH)D2-1a-hydroxylase activity is directly related to serum 25(OH)D levels. Most of the biological effects of 1,25(OH)D2 are due to interaction with BYN, expressed on the cellular and nuclear membranes of various cells of the epidermis, hematopoiesis and immune system [9, 10]. The best indicator for assessing vitamin D status is its active component, serum 25-hydroxyvitamin D. Consensus regarding the optimal level of 25(OH)D in serum has not yet been reached. According to most experts, vitamin D deficiency corresponds to a serum level of <20 ng/ml (Table 1). A level of 12–20 ng/ml is considered insufficient because it is not sufficient for bones and does not correspond to the concept of "full health." Vitamin D deficiency leads to the development of secondary hyperparathyroidism, which leads to loss of bone mineral density (BMD). The serum 25(OH)D level required to maintain normal parathyroid hormone (PTH) concentrations should be >75 nmol/L [11]. According to WHO, vitamin D deficiency occurs in approximately 50% of the world's population, i.e., about 1 billion people worldwide of all nationalities and all age groups suffer from hypovitaminosis D [12, 13]. This "pandemic" is mainly due to lifestyle (long stays indoors) and environmental factors (air pollution, etc.), which reduce exposure to sunlight, which is necessary for the production of vitamin D in the skin under the influence of UV rays. . This problem is of great social importance, since convincing evidence has been obtained of the possible role of vitamin D in the development of malignant neoplasms, cardiovascular pathology, autoimmune diseases, influenza, type 2 diabetes mellitus (DM) and depression [14].

The connection between hypovitaminosis D and diseases such as rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), systemic scleroderma, ankylosing spondylitis, Behcet's disease. Graves' disease, fibromyalgia, multiple sclerosis, allergic rhinitis and asthma in children, Graves' disease have been proven [15]. It has been established that hypovitaminosis D is an independent risk factor for overall mortality in the population [16, 17]. The first study of vitamin D in SLE appeared in 1995, and by 2013 about a hundred such studies had been published [18]. Most of them were mainly concerned with the frequency of hypovitaminosis in SLE, the relationship of vitamin D levels with indicators of activity and damage [19, 20]. However, the question of the effect of vitamin D on the course and prognosis in patients with SLE remains open. According to modern data, vitamin D deficiency occurs in 2/3, and its deficiency occurs in every 5th SLE patient [21]. It is well known that insolation is the most important trigger for SLE. Avoidance of sun exposure and the use of sunscreen are a component of treatment for SLE, so it is not surprising that vitamin D deficiency is present in this disease. Other important factors contributing to the development of hypovitaminosis D in SLE are renal failure, long-term use of certain drugs (glucocorticoids - GCs, antiepileptics). drugs), as well as the presence of antibodies to vitamin D, which increase its clearance [22,23,24].]. The literature provides conflicting information about the relationship between SLE and vitamin D (Table 2). D.L. Kamen et al [25] found a high incidence of vitamin D deficiency in 123 patients with short-duration SLE compared with controls (n=140). Overall, 67% of SLE patients were vitamin D deficient, with mean levels significantly lower in African Americans (16 ng/mL) compared to Caucasians (31 ng/mL). Critically low levels of vitamin D (<10 ng/ml) were found in 22 patients with SLE, mainly in patients with kidney damage and photosensitivity. Similar changes have been observed in patients with a long history of SLE. AM Husiman et al studied vitamin D status (hydroxyvitamin, dihydroxyvitamin and PTH levels) in 25 patients with SLE and 25 patients with fibromyalgia. Hypovitaminosis D was registered in every second patient in both groups, however, statistical differences in the hormonal profile in patients with SLE and fibromyalgia were found. At

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the same time, there is a clear connection with indicators of SLE activity. was discovered by M. Mandai et al from 129 Indian patients[26,27,28]. A feature of this group of patients was the high percentage (79 of 129) of patients at the onset of the disease who were not receiving immunosuppressive therapy.

MATERIALS, METHODS AND RESULTS

An inverse relationship was established between the level of 25(OH)D and disease activity on the SLEDAI scale (r = - 0.42), the level of antibodies to dsDNA (r = - 0.39), the level of α-interferon - IFN (r = -0 ,43) and α-IFN gene expression (r=-0.45) regardless of treatment. The main source of α-IFN in SLE patients is activated by dendritic cells. In vitro studies have demonstrated the ability of vitamin D to inhibit dendritic cell maturation/activation and IFN production; therefore, the negative correlation found between IFN content and 25(Off)D may reflect the participation of this vitamin in the development and activation of SLE. A high incidence of hypovitaminosis D was noted by G. Ruiz-Irastorosa et al. Vitamin D deficiency and insufficiency were present in 75 and 15% of SLE patients, respectively. Higher 25(OH)D levels were reported in female patients receiving aminoquinoline drugs (AQs) and calcium supplements with vitamin D. Photosensitivity was a predictor of vitamin D deficiency, and photoprotection was a predictor of deficiency. Patients were advised to take vitamin D orally. This recommendation was followed by 75% of them: the average level of vitamin D increased significantly, but in most cases did not reach the optimal level. The increase in dihydroxyvitamin D[29,30] levels in the blood serum of SLE patients was not accompanied by a decrease in activity and damage determined by standard methods, but a significant decrease in weakness was recorded.

CONCLUSION

Thus, there is a direct connection between low levels of vitamin D and the occurrence and activation of SLE cannot be excluded, although the development of hypovitaminosis D as a result cannot be denied the disease itself. To assess vitamin status, it is necessary to determine the level of 25(OH)D in the blood serum, and a decrease in this indicator <20 ng/ml (50 nmol/l) is regarded as hypovitaminosis B. In 2011, the US Institute of Medicine formulated recommendations indicating the need to take 400 to 600 IU of vitamin D per day for people aged 1 to 70 years and 800 IU for people over 70. There is no consensus on the tactics of using vitamin D for rheumatic diseases. Several studies have shown that higher doses of vitamin D (>800 IU/day) may be appropriate for patients with RA to achieve optimal levels of this hormone. Data on the effectiveness of vitamin D in patients with SLE. Experimental studies show increased survival and decreased proteinuria in mice with vitamin B supplementation. About 30 years ago, it was found that the use of dihydroxyvitamin D in experimental models of lupus resulted in regression of alopecia, decreased proteinuria, and decreased levels of anti-dsDNA antibodies. Administration of 1,25(OH)D/ before the onset of symptoms may even have a preventive effect against the development of the disease. It was also noted that in mice with spontaneous development of lupus-like disease, the effectiveness of prophylactic administration of vitamin D was comparable to that of high-dose GC. The beneficial effects of vitamin D agonists in preventing disease severity and symptom severity have been demonstrated in mouse models of collagen-induced arthritis. Experimental models of autoimmune encephalomyelitis (a prototype of multiple sclerosis) have shown the protective effect of 1,25(OH)D e analogues on the development of the disease, especially when used together with other immunosuppressants.

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