A CLINICAL CASE BASED ON THE EXPERIENCE OF TREATING HYPERTENSION IN A PATIENT WITH TYPE 2 DIABETES MELLITUS, OBESITY AND VITAMIN D DEFICIENCY

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ABSTRACT

The study of the effect of vitamin D deficiency on the human body is attracting increasing attention from scientists. According to numerous studies, vitamin D deficiency is registered in half of the world's population; among older people this figure reaches 80–90%. At the same time, vitamin D deficiency in men is most often detected in obesity, androgen deficiency, infertility and prostate diseases.

Keywords: vitamin D, obesity, apoptosis, ergocalciferol, cholecalciferol, lumisterol, dihydrotachysterol.

Relevance.

Research in recent years has significantly expanded the understanding of the spectrum of biological activity of vitamin D on human health. This vitamin hormone is currently considered as a regulator of the transcriptional activity of genes that control the mineral homeostasis of bone tissue and blood, the functions of the immune system, apoptosis processes, regulation of blood pressure and other metabolic processes. The formation of the final, hormonally active form of vitamin D is a rather complex process and depends on the supply of the primary substrate and the metabolism of intermediate forms. Violation at any level leads to a decrease in the content and activity of vitamin D, which is usually referred to as "vitamin D deficiency or deficiency." The results of studies conducted in different countries indicate that about 1 billion people in the world have vitamin D deficiency [1–4]. Since most of the biological effects of vitamin D are realized at the genomic level, often changes in health that occur due to its deficiency are not always directly associated with vitamin D. Currently, a number of diseases or pathological conditions associated with vitamin D deficiency have been identified. These include osteoporosis and rickets, diseases of the cardiovascular system, some types of cancer, type 1 diabetes mellitus, damage to nervous tissue and some others [4–9]. In many countries, vitamin D deficiency is considered a "silent epidemic" that has

serious medical and medical-social consequences and requires adequate correction [10–13]. Vitamin D is a steroid whose hydroxylated derivatives have hormonal activity. In the literature, the term “vitamin D” often refers to functionally low-active forms that are similar in chemical structure (ergocalciferol, cholecalciferol, lumisterol, dihydrotachysterol, and others). However, based on biological effects (enzyme-mediated step-by-step synthesis of the active substrate, long-term mechanism of action, interaction with specific nuclear receptors), the final product of vitamin D metabolism - calcitriol - can be considered a true hormone. The formation of hormonally active vitamin D derivatives in the human body is carried out through a two-step synthesis from endogenous and exogenous precursors. The contribution of the exogenous pathway of vitamin D provision is small and amounts to only 20–30%. With food (salmon, tuna, cod, beef liver, butter, milk, cheeses, egg yolks, some mushrooms and cereals and other products), the exogenous precursor - ergocalciferol (vitamin D2) enters from the digestive tract into the lymphatic and then into the circulatory system . Of much greater importance is the endogenous pathway for the formation of vitamin D, which consists in the synthesis of the endogenous precursor cholecalciferol (vitamin D3) from the 7-dehydrocholesterol located in the dermal layer of the skin under the influence of short-wave ultraviolet irradiation of the B spectrum (wavelength 290–315 nm), which also enters the systemic circulation [1, 8, 12]. The presence of skin pigmentation, as well as the use of closed clothing and sunscreens, help reduce the production of vitamin D3 in the skin. In addition, age, the use of certain medications, the amount of adipose tissue in the body, and malabsorption syndrome have a negative impact on the exogenous supply and endogenous formation of vitamin D in the human body [4, 12, 14]. The metabolism of vitamin D (from food or synthesized in the skin) consists of step-by-step enzyme-mediated hydroxylation with the formation of the final, most active hormone calcitriol (1,25(OH)2 D3) and the less active form 24,25(OH)2 D3. The first stage of hydroxylation occurs in the liver and depends on the amount of the initial substrate, is controlled by the enzyme 25-hydroxylase and ends with the formation of the intermediate form 25(OH)D - calcidiol. The formation of calcidiol depends on the functional state of the liver and in case of its impairment (liver failure) can be reduced. Extracellular transport of vitamin D and its metabolites is carried out using vitamin D-binding globulin, lipoproteins and albumins, changes in the concentration of which can lead to changes in vitamin D status. Partial 25(OH)D is deposited in adipose and muscle tissue, but is mainly transported by the bloodstream to kidneys to the second stage of hydroxylation. The presence of obesity can lead to an increase in the volume of vitamin D depot and reduce the concentration of circulating calcidiol in the blood. The second stage of hydroxylation occurs mainly in the proximal renal tubules and extrarenally (skin cells, monocytes, placenta, bone, cells of the

immune system and some other tissues) under the influence of the enzyme 1α-hydroxylase (CYP27B1), ending with the synthesis of calcitriol. The completion of vitamin D metabolism is carried out under the influence of the enzyme 24-hydroxylase (CYP24), which catalyzes the process of transition of 1,25(OH)2 D3 into water-soluble biologically inactive calcitroic acid, excreted in bile [4, 7]. The formation of calcitriol is regulated by a complex of endogenous and exogenous factors. The main stimulator of 1,25(OH)2 D3 synthesis in the kidneys is parathyroid hormone (PTH), the level of which in the blood plasma is influenced by the "feedback" principle by both the concentration of 1,25(OH)2 D3 itself and the content of calcium and phosphorus . In addition, a number of hormones have a stimulating effect: androgens, estrogens, calcitonin, prolactin and growth hormone. Inhibitors of 1,25(OH)2 D3 synthesis include corticosteroids, synthetic analogues of calcitriol, some growth factors (for example, fibroblast growth factor FGF23) and drugs (glucocorticoids, anticonvulsants, and others) [1, 7–8]. In the presence of kidney diseases leading to the development of nephrotic syndrome, an increase in urinary excretion of calcitriol has been proven, and in the case of chronic renal failure, a decrease in its formation [1, 14]. 1,25-(OH)2 D, like other steroid hormones, it binds and activates its receptors (VDR), which belong to the nuclear class and act as transcription factors that control the expression of a large number of genes. Data have been obtained on the presence of vitamin D receptors in more than 40 tissues, during interaction with which 3–5% of the human genome is controlled [7–8, 15]. Only some cells, such as red blood cells, uterine smooth muscle cells, and Purkinje cells of the brain, do not have receptors for vitamin D [8]. With a decrease in VDR activity, which is often caused by genetic factors, the post-receptor effects of 1,25(OH)2 D3 are disrupted, which in turn leads to hypocalcemia due to a decrease in intestinal absorption and renal reabsorption of calcium, an increase in the production of parathyroid hormone and the development of secondary hyperparathyroidism [7– 8].

Target: to evaluate the effect of correction of vitamin D deficiency on the dynamics of blood pressure in a male patient with hypertension, type 2 diabetes mellitus, obesity and vitamin D deficiency.

MATERIALS AND METHODS

patient P., 50 years old, complained of headache, periodic dry mouth and frequent urination, weakness, fatigue, increased blood pressure to 155/95 mmHg. From the medical history - diabetes mellitus (DM) type 2 and hypertension (HD) for 5 years, age-related hypogonadism (AH) and vitamin D deficiency have been established over the past 6 months. On examination, the patient is well-nourished, height 184 cm, weight 139 kg, BMI 41 kg/m2, WC-130 cm. The thyroid gland is not palpable. In the heart: the sounds are muffled, the rhythm is correct, blood pressure is 155/95 mmHg,

heart rate is 85 beats/min. Peripheral pulsation in the arteries of the feet is preserved. In addition to the general clinical examination, the patient had systolic and diastolic blood pressure (SBP) and (DBP) measured, respectively, body mass index (BMI) was determined using Quetelet's formula, and waist circumference (WC) was measured. Carbohydrate metabolism was assessed by the glycotriad - glycemia on an empty stomach and 2 hours after a meal, as well as by the level of glycated hemoglobin A1c. Analysis of fat metabolism was carried out using the lipid spectrum - TC, HDL, LDL, TG, CA. Hormonal examination included determination of follicle-stimulating and luteinizing hormones (FSH) and (LH), respectively, total testosterone (T), prolactin, thyroid-stimulating hormone (TSH). Vitamin D levels were determined. For the treatment of diabetes, the patient was prescribed metformin long 1500 mg and vildagliptin 100 mg per day; for the treatment of hypertension - valsacor 80 mg and nevibolol 5 mg per day; for the treatment of CH - low-dose testosterone 1% 50 mg per day and for the correction of vitamin D deficiency - cholecalceferol 10 drops per day.

RESULTS

After 6 months of therapy, in addition to subjective improvement in condition, disappearance of thirst and dry mouth, cessation of headaches, improvement in performance and general tone, the patient's clinical and metabolic indicators also significantly improved. The patient's weight decreased by 21% to 110 kg, BMI decreased by 20% to 33.3 kg/m2, WC decreased by 16.9% and amounted to 108 cm. Blood pressure indicators reached target values - SBP and DBP decreased by 18.9 and 10%, respectively , and amounted to 130 and 85 mmHg.

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