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# STATE OF HORMONAL AND CALCIUM-PHOSPHORUS METABOLISM IN PATIENTS UNDERGOING THYROID SURGERY

**Atayeva Dilafruz**
Teacher of public health technical college named after Republic No. 1 Abu Ali Ibn Sina

**Annotation:** In this paper we propose the study of hormonal disorders and calcium-phosphorus metabolism, resulting from surgical treatment of diseases of the thyroid gland. The thyroid gland is an organ of the endocrine system and in addition to other functions, performs the function of supporting calcium - phosphorus homeostasis in the body. During operations on the thyroid gland is a violation of the hormonal balance, in consequence of the violation of calcium-phosphorus homeostasis. The issue presented in the medical literature, but it is not fully understood, and a definite opinion about this no.

**Key words**: Thyroid gland, calcium and phosphorus metabolism, calcitonin, parathyroid hormone, triiodothyronine

The thyroid gland is one of the organs of the endocrine system, and one of its functions is to support the metabolism of calcium and phosphorus in the body. Parathyroid hormones, the active form of vitamin D1,25(OH)2, thyrocalcitonin participate in the regulation of bone metabolism, in addition, triiodothyronine, thyroxine and thyroid-stimulating hormone play a certain role in the regulation of bone metabolism. Calcium receptors are located in many tissues, such as the parathyroid glands, C-cells of the thyroid gland, kidneys, pituitary gland, brain, bone marrow, intestines, skin, etc. It is generally accepted that parathyroid hormone is the main regulator of bone mineral density, its level depends on the concentration of calcium in the blood serum. Thyrocalcitonin is an antagonist of parathyroid hormone, the existence of which was first proposed in 1961. Later, it was found that thyrocalcitonin is synthesized by C-cells of the thyroid gland. However, there is evidence that these cells are not the only place where thyrocalcitonin is produced. Hargis et al. (1966), A.A. Bulatov (1970) discovered thyrocalcitonin activity in the cytoplasm of all thyroid cells that secrete thyroglobulin. It is generally accepted that parathyroid hormone and thyrocalcitonin are antagonists and the main property of thyrocalcitonin is its ability to reduce the level of Ca in the blood serum. But there are some contradictions about the role of thyrocalcitonin in the process of bone metabolism. According to N.V. Danilov and V.G. Aristarkhov (2014), surgical intervention on the thyroid gland affects the mineral density of the bone system. There is a direct relationship between the volume of the operation and the degree of bone mineral density disorders. However, S.M. Cherenko (2011) believes that the role of calcitonin is not completely clear, since the effect it has is minimal and is not revealed by other mechanisms. Remodeling is the main process that maintains bone density at a certain level, consists of bone resorption and bone formation, which is supported by antagonist cells osteoblasts and osteoclasts. The

works of various researchers have shown a reliable increase in the number of patients with hip fractures suffering from thyrotoxicosis, compared with healthy people, and the risk of fractures increased with age. There is no doubt that thyroid hormones stimulate osteoblasts and osteoclasts, but for a long time it was believed that their effect on these cells is not direct. In vitro studies have shown that the presence of osteoblasts is necessary to stimulate osteoclasts, the mechanism of this relationship remains a mystery. It was found a little later that triiodothyronine exerts its effect on osteoblasts directly through alpha receptors (TRα) in thyroid cells, or by influencing the synthesis of alkaline phosphatase, collagen I and osteocalcin. Triiodothyronine also changes the synthesis of receptors to parathyroid hormone and thereby regulates the response of osteoblasts to it. Stimulation of cytokines (interleukin-6, interleukin-8, prostaglandin E2) involved in osteoclastogenesis plays an important role in the interaction of triiodothyronine and osteoclasts [9, 10]. In the absence of estrogens, increased bone loss occurs in patients with elevated levels of thyroid hormones, which demonstrates the protective effect of estrogens on bone tissue. Estrogens increase OPG, suppress the synthesis of tumor necrosis factor α (TNFα), interleukins-1, 6 and macrophage colony-stimulating factor, which serves as a mechanism for reducing osteoclastogenesis. Thyroid surgery causes disruption of hormone production and regulation of calcium phosphorus metabolism. Most often, a decrease or absence of thyrocalcitonin and an increase in parathyroid hormone are observed. The level of parathyroid hormone may decrease after thyroid surgery, the reason for this is damage to the parathyroid glands themselves or the vessels that feed them. The purpose of the study: to study changes in the indicators of some thyroid hormones in patients operated on for goiter.

The study group consisted of 50 women suffering from goiter, undergoing planned surgical treatment in the surgery department of RKB №4 in Saransk, their age ranged from 36 to 64 years. Nodular goiter was present in 10 (20%) patients, relapse of nodular goiter was observed in 5 (10%) patients, diffuse-nodular goiter - in 35 (70%). The subjects underwent surgical intervention: GTE - 30%, STC - 62%, TE - 8%. The control group consisted of 10 healthy women aged 30 to 55 years. To study the hormonal status and calcium-phosphorus metabolism, blood was taken from patients before and after surgery on the third day. Blood centrifugation was performed to obtain plasma. Plasma Ca and P levels, parathyroid hormone, calcitonin, and T3 were determined using the enzyme immunoassay method on a Stat-Fax apparatus. At different stages of the analysis, the following were used: a 2100 microplate photometer with a wavelength of 405; 450; 492; 630 nm, a Stat-Fax – 2200 microplate incubator-shaker, and a StatFax 2600 microplate washer. An ultrasound examination of the thyroid tissue volume before and after surgery was performed on a TOSHIBA apparatus. Results and discussion. The Ca level significantly ($p<0.01$) decreased in all patients after surgery. The degree of decrease did not depend on the volume of the thyroid remnant. Phosphorus increased after surgery; no reliable relationship was found between changes in phosphorus levels and the volume of the thyroid remnant. Based on the study data (table), it is evident that the calcitonin level in

the blood after thyroidectomy was not determined. After thyroid resection, the calcitonin level in the blood was distributed as follows: in the GTE group, the calcitonin level after surgery was higher than in the STC group, after GTE, the calcitonin level changed less than after STC. Thus, it can be argued that the calcitonin level depends on the volume of the thyroid remnant after thyroid surgery.

Despite the fact that calcitonin and parathyroid hormone are antagonist hormones, an increase in parathyroid hormone in response to a decrease in calcitonin is not observed, which may be associated with damage to the parathyroid glands during surgery. There is a decrease in the level of parathyroid hormone in patients with all types of surgical intervention (table), the degree of decrease in parathyroid hormone does not depend on the volume of the operation. The level of T3 in the blood after thyroidectomy is not determined, which is a consequence of the absence of thyroid tissue after surgery. There is a dependence of the degree of T3 decrease on the volume of the thyroid remnant after thyroid resections, the maximum T3 decrease is with STC, the minimum with GTE.

1. The level of calcitonin and T3 in the blood changes after thyroid surgery depending on the volume of the operation.

2. A preliminary study showed that the degree of decrease in Ca and P in the blood is not associated with the choice of surgery.

3. A decrease in the level of parathyroid hormone is observed in all patients who underwent thyroid surgery. However, the data obtained do not make it possible to fully determine the mechanisms of thyroid involvement in the regulation of bone metabolism, the role of surgical interventions in the development of osteoporosis and, in general, in the disruption of phosphorus-calcium metabolism in the body.

Many questions remain regarding the mechanism of action of thyroid hormones on bone metabolism. The opinion on the role of thyrocalcitonin and TSH in bone metabolism disorders in thyroid diseases is ambiguous. Research is ongoing, the results of which will help to reveal the mechanisms of bone disorders in thyroid pathology and its surgical treatment and can be a theoretical basis for postoperative rehabilitation of such patients.

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