STATE OF BONE METABOLISM IN THYROID DISEASES.

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Annotation: This paper discusses the relationship between thyroid diseases and bone metabolism. Thyroid hormones play a crucial role in the regulation of bone remodeling processes, affecting both bone formation and resorption. Disorders such as hyperthyroidism and hypothyroidism significantly influence bone mineral density and may lead to increased fracture risk. The study highlights the pathophysiological mechanisms linking thyroid dysfunction to skeletal health and emphasizes the importance of early diagnosis and management of bone-related complications in patients with thyroid diseases.
Keywords: Thyroid diseases, bone metabolism, hyperthyroidism, hypothyroidism, bone mineral density, osteoporosis, skeletal health, hormone imbalance.

Osteopathy is a common manifestation of endocrine pathology. In 1891, Recklinghauser first described multiple fractures in a patient with untreated thyrotoxicosis. Bone metabolism is regulated by parathyroid hormone produced by the parathyroid glands, the active form of vitamin D1,25(OH)2 (vitamin D3), thyrocalcitonin synthesized by C-cells of the thyroid gland, T3, T4 and thyroid stimulating hormone (TSH) can also participate in this process. The main regulator of bone mineral density (BMD) is parathyroid hormone (PG), the level of which depends on the concentration of calcium in the blood. Bone tissue is the main depot of calcium and phosphorus in the body and, in addition to the mechanical function, performs a metabolic function, maintains calcium-phosphorus homeostasis. Calcium receptors are located in many tissues, such as the parathyroid glands, kidneys, thyroid C-cells, brain, intestines, pituitary gland, bone marrow, skin, etc. Bone tissue is quite variable and subject to remodeling, which consists of two processes: bone resorption and bone formation, with the first, bone mineral density decreases, with the second, it increases. The remodeling process allows bones to adapt to loads and maintain strength. The remodeling process is supported by two types of cells - osteoblasts and osteoclasts, the work of these cells is closely related to each other, and they play opposite roles in the remodeling process, osteoclasts locally remove old bone tissue, and osteoblasts are engaged in increasing bone mass. Stimulators of the remodeling cells are most likely paracrine factors, they are released by osteocytes when exposed to physical stress. Destruction of the protein matrix of bone tissue is carried out by osteoclasts at low pH with the help of hydrogen ions and lysosomal enzymes; after performing their function, osteoclasts undergo apoptosis. The second phase of bone remodeling is osteogenesis, in which osteoblasts form a bone matrix in the areas of resorption. In case of imbalance of these two mechanisms, osteopathies and osteoporosis occur, and the resorptive surface is not completely filled with bone matrix. The antagonist of PG is thyrocalcitonin, the

existence of which was first suggested in 1961. Subsequently, it was revealed that thyrocalcitonin is synthesized by C-cells (parafollicular cells) of the thyroid gland. However, these cells are apparently not the only place where thyrocalcitonin is produced. Thus, Hargis et al. (1966), A.A. Bulatov (1970) discovered thyrocalcitonin activity in the cytoplasm of all thyroid cells that secrete thyroglobulin. The hormone thyrocalcitonin is a polypeptide with a molecular weight of 3000–8700. In contrast to parathyroid hormone, the main property of thyrocalcitonin is its ability to reduce the level of calcium in the blood serum. Hyperthyroidism and hypothyroidism in thyroid diseases affect calcium-phosphorus metabolism and bone metabolism. Thyroid hormones affect both bone formation and bone resorption. Thyrotoxicosis is accompanied by increased bone remodeling and resorption, with the latter predominating, which in turn leads to a loss of 10% of bone mass per cycle of bone remodeling and the development of osteoporosis. The reason for this phenomenon is the increased activity of osteoclasts and osteoblasts, acceleration of their work and reduction of the duration of bone remodeling cycles. The level of bone metabolism markers increases. The works of various scientists 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 proportionally to age.

Thyrotoxicosis is often accompanied by hypocalcemia, hyperphosphatemia, hypercalciuria. Moderate increase in blood Ca levels is observed in 20% of patients. According to various authors, changes in calcium-phosphorus metabolism in thyrotoxicosis depend on reproductive status. Most authors believe that calcium in the blood decreases mainly in women suffering from postmenopausal thyrotoxicosis. However, more modern studies show that hypo- or normocalcemia, hyperphosphatemia, increased calcium excretion in the urine, as well as increased PTH levels are more common in young women. These indicators positively correlate with bone mineral density. After eliminating thyrotoxicosis, restoration of lost bone mineral density occurs in patients of reproductive age, but does not occur in women in the postmenopausal phase and requires appropriate prevention and treatment. According to H. Nielsen, complete restoration of bone mineral density occurs with normalization of thyroid function after two years. Thyrotoxicosis is also one of the risk factors for the development of osteoporosis in postmenopause.

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