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IMPAIRMENT OF FUNCTIONS ORGANS AND SYSTEMS IN HYPOPARETHYROIS

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Abstract:

Thyroid hormones (thyroid hormones) are necessary for the normal functioning of the kidneys, which, in turn, serve not only as an organ for the metabolism and excretion of thyroid hormones, but also as a target for the action of some thyroid hormones. Hypothyroidism and hyperthyroidism are associated with clinically significant changes in renal function. Hypothyroidism is accompanied by a decrease in glomerular filtration rate (GFT), a change in the kidneys' ability to remove water from the body.

Keywords: Hypoparathyroidism, parathyroid hormone, parathyroid glands, kidney damage

Hypoparathyroidism is a condition characterized by a decrease in the production of parathyroid hormone (PTG) and tissue resistance to its effects, which is accompanied by a violation of phosphorus-calcium metabolism. PTG helps maintain mineral homeostasis and increases reabsorption in the medullary cells of the renal distal tubules and distal convoluted tubules. Chronic magnesium deficiency leads to the development of hypocalcemia by decreasing PTG secretion and increasing resistance to its effects [1,2,3,4,5].

In addition, calcitriol controls the transfer of calcium ions from epithelial cells to the blood, due to which their concentration in the extracellular fluid is maintained at the level necessary for mineralization of the organic matrix of bone tissue. In the kidneys, calcitriol stimulates the reabsorption of calcium and phosphate ions. Along with PTG and calcitriol, calcitonin synthesized by parafollicular S-cells (TG) of the thyroid gland is involved in the control of calcium metabolism. Its secretion increases with an increase in the concentration of calcium in the blood serum and, on the contrary, decreases with a decrease.

As a PTG antagonist, calcitonin acts on the skeleton, where inhibition of bone resorption results in reduced calcium release. In addition, it reduces the tubular reabsorption of calcium ions in the kidneys, thereby stimulating their excretion by the kidneys in the urine. Calcitonin's main physiological role is to support growth and maintain bone structure during pregnancy and lactation when the demand for calcium in tissues increases dramatically [3,4,5].

Thyroid hormones (thyroid hormones) are necessary for the normal functioning of the kidneys, which, in turn, serve not only as an organ for the metabolism and excretion of thyroid hormones, but also as a target for the action of some thyroid hormones. Hypothyroidism and hyperthyroidism are associated with clinically significant changes in renal function. Hypothyroidism is accompanied by a decrease in glomerular filtration rate (GFT), a change in the kidneys' ability to remove water from the body. An increase in thyroid hormone levels leads to an increase in GFT and renal blood flow. Kidney failure affects the work of the thyroid gland. Chronic kidney disease is accompanied by significant effects on the hypothalamic-pituitary-thyroid axis. There is a connection between thyroid cancer and kidney tumors. Recent studies show that thyroid hormones, especially T3, can be considered as a marker of survival in patients with kidney disease.

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Hypothyroidism is the most common disorder of thyroid function. Since thyroid hormones are involved in the regulation of metabolism, the differentiation and functioning of body tissues, hypothyroidism can cause changes in all organs and systems.

Hypothyroidism is a clinical syndrome caused by the hypofunction of the thyroid gland and characterized by a decrease in the amount of thyroid hormones in the blood serum [1]. The prevalence of primary manifest hypothyroidism in the population is 0.2-2%, subclinical - up to 10%. This disease is associated with high total and cardiovascular mortality [2].

The most common cause of primary hypothyroidism is chronic autoimmune thyroiditis. In rare cases, the disease develops during the treatment of thyrotoxicosis. Clinical manifestations of hypothyroidism are different, because almost all organs and systems are affected. Deficiency of thyroid hormones is associated with neonatal respiratory distress syndrome - thyroid hormones are involved in the production of surfactants and development of fetal lungs.

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Urinary system. Hypothyroidism directly or indirectly affects kidney function. Secondary arterial hypertension due to a decrease in systolic volume and an increase in vascular resistance leads to a decrease in renal perfusion, filtration capacity, and glomerular filtration rate (GFT). Patients also have decreased renal tubular reabsorption, increased sodium excretion, and decreased potassium excretion. Plasma renin activity, aldosterone level may decrease.

These indicators normalize during replacement therapy with sodium levothyroxine [5]. Hyponatremia develops in some cases due to a decrease in the primary flow of urine to the renal tubule system [1].

An increase in serum creatinine is observed, which normalizes after compensation of hypothyroidism. In patients with chronic kidney disease, hypothyroidism impairs kidney function. Therefore, when there is an unexplained sharp decrease in GFT, it is recommended to evaluate the level of thyroid-stimulating hormone.

Myoglobin and creatine kinase levels are elevated in patients due to myopathy and decreased clearance. Cases of rhabdomyolysis and acute renal failure have been described [6]. Hypothyroidism is characterized by a disorder of purine metabolism can cause hyperuricemia. The latter is detected in 30% of patients with hypothyroidism. 7% of patients develop gout [7].

But kidney pathology can be a consequence not only of thyroid gland dysfunction, but also of autoimmune nephropathy and proteinuria [8]. When treated with thyroid hormones, the hemorrhagic syndrome is completely reversed, and the level of coagulation factors is also restored.

Hypothalamic-pituitary system. High production of thyrotropin-releasing hormone by the hypothalamus during hypothyroxinemia not only increases the release of thyroid-stimulating hormones by the adenopituitary gland, but also prolactin. In primary hypothyroidism, this can lead to the development of hyperprolactinemia. Manifested by hypogonadism, hyperprolactinemia, hypogonadism, oligo-opsomenorrhoea or amenorrhoea, galactorrhea, secondary polycystic ovary syndrome [16].

Thyroid hormone deficiency affects many physiological functions and metabolic processes in the body, and therefore changes can be observed in all organs and systems. Clinical manifestations of hypothyroidism are very diverse and non-specific.

PTG deficiency leads to hyperphosphatemia, which directly leads to increased renal convoluted tubule phosphate reabsorption and indirectly to hypocalcemia [5]. Based on the above points, it can be concluded that mineral metabolism is controlled by the balance of three hormones in the body: PTG, calcitonin and calcitriol. As a result of parathyroid hormone deficiency, an increase in the amount of calcitonin and a decrease in the synthesis of calcitriol develop, and the clinic of hypoparathyroidism occurs. The correct interpretation of this will make it possible to determine the correct treatment and preventive measures, as well as predict possible complications. Genetic etiology of hypoparathyroidism occurs in less than 10% of cases. The main cause of hypoparathyroidism is chromosomal microelements and monogenic anomalies.

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