

HYPOTHYROIDISM - ENDOCRINE AND METABOLIC DISORDERS.

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Abstract: Hypothyroidism is a deficiency of thyroid hormones. Symptoms include cold intolerance, general weakness, and weight gain. Signs may include a typical appearance and facial expression, hoarse slow speech, and dry skin. Diagnosis is carried out using tests for thyroid function. Treatment includes the administration of thyroxine.

Keywords: thyroxine, thyrostatic drug therapy, hoarse slow speech.

Hypothyroidism is a disease caused by a decrease in the function of the thyroid gland. The edematous form of the disease is called myxedema (mucosal edema). There are primary, secondary and tertiary hypothyroidism. In primary (thyrogenic) hypothyroidism, the place of localization of the pathological process is the thyroid gland, in secondary – the pituitary gland, in tertiary – the hypothalamus. Causes of primary hypothyroidism: thyroid abnormalities; endemic goiter and cretinism, inflammatory diseases of the thyroid gland, surgical removal of the thyroid gland, treatment with radioactive iodine, thyrostatic drug therapy, cancer metastases to the thyroid gland, chronic infections tuberculosis, syphilis, violation of thyroid hormone biosynthesis (congenital defects of enzyme systems, lack of yod. Secondary hypothyroidism is a consequence of diseases of the pituitary gland with a decrease in its functions. There may be an isolated insufficiency of thyroid-stimulating hormone (congenital and acquired) or the secretion of biologically inactive TSH.

Tertiary hypothyroidism is caused by a primary lesion of the hypothalamic centers secreting thyrotropin-releasing hormone (tyroliberin). The development of hypothyroidism is based on a long and pronounced deficiency of the specific action of thyroid hormones with a decrease in oxidative processes and thermogenesis. With a decrease in the amount of thyroid hormones, the metabolism in the body is disrupted, the work of the cardiovascular system, gastrointestinal tract, mental and sexual activity deteriorates. Symptoms of hypothyroidism develop slowly, imperceptibly, so patients do not consult a doctor for a long time. With the disease, there are: lethargy, drowsiness, deterioration of memory, attention, mental activity, intolerance to cold and heat, dry skin, hair loss, swelling, weight gain, constipation, menstrual disorders in women, decreased potency and sexual desire in men. Primary hypothyroidism is caused by a decrease in the secretion of T4 and T3 by the thyroid gland. Low serum levels of T4 and T3 and increased levels of thyroid-stimulating hormone (TSH). The most common cause in the United States is autoimmune disorders. Hashimoto's thyroiditis usually leads to primary

hypothyroidism, often associated with a dense goiter or – in the later stages – with fibrosis of the thyroid gland, accompanied by its atrophy and a decrease or complete loss of function.

The second most common cause is the development of post-therapeutic hypothyroidism, especially post-radiation therapy or surgical treatment of hyperthyroidism. Hypothyroidism, which develops with an overdose of propylthiouracil, thiamazole and iodine, usually passes after discontinuation of drug treatment. Iodine deficiency can cause endemic goiter and goiter hypothyroidism. Most patients with goiter, the cause of which is not Hashimoto's thyroiditis, have an euthyroid or hyperthyroid nature of the disease. With iodine deficiency, the homogeneous structure of the thyroid gland is disrupted. In response, TSH is released, which increases the size of the thyroid gland and its ability to absorb iodine; as a result, goiter develops. In the case of acute iodine deficiency, the patient develops symptoms of hypothyroidism, which is rare after the introduction of iodized salt in the United States.

With iodine deficiency, congenital hypothyroidism occurs. Severe iodine deficiency in some regions, leading to congenital hypothyroidism (formerly called endemic cretinism), is the main cause of mental retardation worldwide. In rare cases, goiter hypothyroidism is caused by congenital defects of thyroid hormone synthesis enzymes. Hypothyroidism occurs in patients taking lithium (which probably inhibits the secretion of thyroid hormones). Hypothyroidism can also be observed in patients taking amiodarone or other iodine-containing drugs, in patients taking interferon alpha, and in patients taking checkpoint inhibitors or some antitumor tyrosine kinase inhibitors. Hypothyroidism may result from radiation therapy for laryngeal cancer or Hodgkin's lymphoma. Persistent hypothyroidism often develops after radiation therapy, and thyroid function (determination of serum TSH levels) in such patients, it is necessary to evaluate every 6-12 months.

Secondary hypothyroidism.

Secondary hypothyroidism is a consequence of insufficient production of thyrotropin-releasing hormone (TRH) by the hypothalamus or insufficient production of TSH by the pituitary gland. Sometimes a decrease in TSH secretion associated with a deficiency of TRH is called tertiary hypothyroidism.

Subclinical hypothyroidism.

Subclinical hypothyroidism is diagnosed with elevated serum TSH levels in patients with minimal symptoms of hypothyroidism (or their complete absence) and normal serum levels of free thyroxine (T4). Subclinical thyroid dysfunction is relatively common – in

about 15% of older women and 10% of older men – especially with Hashimoto's thyroiditis. If the serum TSH level is > 10 iU/l, there is a high probability of developing obvious hypothyroidism with a decrease in the level of free T4 over the next 10 years. Such patients are also more likely to develop hypercholesterolemia and atherosclerosis. Levothyroxine should be prescribed even in the absence of symptoms. In patients with TSH levels between 4.5 and 10 iU / l and symptoms of early hypothyroidism (for example, fatigue, depression), trial therapy with levothyroxine is performed.

Levothyroxine treatment is also indicated for pregnant or planning pregnancy women to prevent the adverse effects of hypothyroidism on the course of pregnancy and fetal development. Patients should annually check the level of TSH and free T4 in the blood serum to assess the progression of the disease in the absence of treatment or in order to adjust the dose of levothyroxine. To prevent hypothyroidism, it is very important to eat foods rich in iodine — lettuce, buckwheat, beetroot, spinach, and most importantly — algae, including kelp (seaweed) and fucuses. As a tonic, various vitamin and nutrient mixtures are useful, the most common of which is a combination of lemons, honey and nuts. Speaking about the prevention of the disease, it is impossible not to touch on the problem of the environment. Of course, her condition does not depend on us in many ways, but it is possible to organize your proper communication with her: to be outdoors more often, try to maintain cleanliness, from time to time to carry out cleansing procedures.

And one more important point: the thyroid gland reacts very much to stress, nervous overstrain, emotional breakdowns and insomnia. It is necessary to use various means for relaxation. With hypothyroidism, foods such as soy, peanuts, millet, cabbage, Brussels sprouts, cauliflower, Brussels sprouts, spinach, turnips, peaches and pears, mustard, radishes, turnips, rutabaga, corn, beans should be avoided. These foods block the use of iodine. You should take vitamins such as: A and beta-carotene, B vitamins, vitamins C, E, D and Zinc. Iodine, the daily norm of which is 150 mcg, is contained in fish (this product should always be on the table of those who care about their health) and iodized salt. The most useful vegetables for the thyroid gland are beets, carrots, potatoes, tomatoes, lettuce and spinach.

Natural sources of iodine:

Iodine contains iodized salt, seafood, sea fish, red and brown algae, fish oil, sea cabbage, dairy products, buckwheat, fresh vegetables and fruits, dried apricots, chicken yolk, liver, potatoes.

Iodine is also found in garlic, onions, mushrooms, sesame seeds, soybeans, asparagus, spinach, pumpkin.

To find out if our body has enough iodine, we need to apply an iodine mesh (on any part of the body) and see how long it will last. If the grid disappears within two hours, there is a deficiency of iodine. Therefore, you should regularly consume products containing iodine: sea fish and seaweed, persimmons, beets, apples (a lot of iodine in apple seeds - in six seeds a daily dose).

When cooking, iodine is better preserved if:

- pour water so that it only covers the contents of the pan;
- when cooking vegetables, lower them whole or coarsely chopped into boiling water, or even better, cook them for a couple, tightly closing the pan with a lid.

At high boiling, 50% of iodine in meat and fish is destroyed; 30% - in vegetables and fruits. Milk loses 25% of the mineral during prolonged boiling. But, whatever it was, the simplest and most effective way to prevent iodine deficiency was the use of iodized salt. Although it should be handled skillfully:

- when heated, the iodine almost completely evaporates and therefore it is better to salt the dish
- not during the cooking process, but immediately before use;
- it is not recommended to use iodized salt for salting or pickling, because
- pickles will ferment or become bitter;
- iodized salt retains its healing properties only for 3-4 months
- after manufacture, so you need to pay attention to the dates printed on the package;
- it is necessary to store such salt according to the rules: if it gets wet or stays in
- an open salt shaker for a long time, the iodine evaporates.

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