TREATMENT OF EUTHYROID GOITTER

Scientific adviser: **Kurbanova Nozima Sabirdjanovna**
Assistant of the Department of Endocrinology, Samarkand State Medical University

[1]Shodieva M., [2]Samizhanova M., [3]Rustamova N.
Students of the Samarkand state Medical University

Keywords. euthyroid goiter, levothyroxine, iodine preparations.

Annotation. The article briefly summarizes modern ideas about the pathogenetic therapy of euthyroid goiter. The advantages, disadvantages and potential effectiveness of monotherapy with iodine preparations, suppressive therapy with levothyroxine preparations and combination therapy with iodine and levothyroxine preparations are considered. The principles of postoperative prevention of goiter recurrence are described[8-13].

Relevance. The most common cause of euthyroid goiter is iodine deficiency, which is determined throughout Uzbekistan. In endemic regions, about 90-95% of cases of enlarged thyroid gland are associated with iodine deficiency; in children - up to 99%. David Marin was the first to scientifically substantiate the connection between iodine deficiency and hyperplastic processes in the thyroid gland at the beginning of the twentieth century[1-7]. He was the first to discover a relationship between the volume of the thyroid gland and its iodine content, and also described the histological changes characteristic of endemic goiter. After the regulation of the thyroid gland by the hypothalamic-pituitary system was described, in particular the negative feedback between the production of TSH and T4, animal experiments showed that artificially simulated severe iodine deficiency leads to an increase in TSH levels and the formation of goiter. In accordance with this, a theory was put forward according to which TSH not only has a specific stimulating effect on the thyroid gland, but also causes its increase, and TSH itself began to be regarded as the main trophic stimulator of the thyroid gland[16-21]. However, according to many studies, in individuals living in regions with normal iodine supply, as well as moderate and mild iodine deficiency, TSH levels do not differ significantly; it increases only with severe iodine deficiency, which is relatively rare[22-24].

Results. Diffuse euthyroid goiter is the most common, but at the same time the most harmless and easily treatable thyroid disease. A small euthyroid goiter in itself does not pose any danger at the time of diagnosis. The danger to human health of

diffuse euthyroid goiter is determined by the risk of further progression of iodine deficiency thyroid pathology. The first stage of progression is a diffuse enlargement of the thyroid gland without disruption of its function. Depending on the severity of iodine deficiency, diffuse goiter can form in 10-80% of the population. Individual thyroid cells turn out to be more sensitive to ARF stimulation, resulting in preferential growth. This is how nodular and multinodular euthyroid goiter is formed. At the next stage, the described compensatory processes acquire pathological significance. In individual actively dividing thyrocytes, reparative processes begin to lag, as a result of which mutations accumulate, among which the so-called activating ones are of greatest clinical importance; as a result of these mutations, daughter cells acquire the ability to autonomously, i.e. outside the regulatory effects of TSH, produce thyroid hormones. The final stage of the natural morphogenesis of iodine deficiency goiter is nodular and multinodular toxic goiter. This process takes many decades, as a result of which nodular and multinodular euthyroid and toxic goiter most often occurs in the elderly. According to many epidemiological studies, one of the most serious problems of mild and moderate iodine deficiency is the high incidence of multinodular and nodular toxic goiter in the older age group.

A well-proven alternative to L-T4 monotherapy in clinical studies is the combined administration of iodide and L-T4. The easiest way to do this is with drugs that simultaneously contain both components - iodine and L-T4 in the most common doses. For example, Iodtirox contains 100 mcg of L-T4 and 100 mcg of potassium iodide in one tablet. This drug immediately affects two main pathogenetic mechanisms of goiter development[25-30]. The main advantages of combination therapy are the faster achievement of a reduction in thyroid volume (due to L-T4) and the leveling of the phenomenon of L-T4 withdrawal (goiter relapse) due to iodide, which prevents the decrease in intrathyroidal iodine content that occurs with L-T4 monotherapy. In principle, L-T4 and iodide can be prescribed sequentially: at the beginning, rapid regression of gland volume is achieved against the background of suppressive therapy with L-T4, followed by the addition of 100-200 mcg of iodide. An argument in favor of sequential rather than simultaneous administration of L-T4 and iodide is the fact that TSH suppression, which develops against the background of L-T4 administration, suppresses the transport of iodine into the thyroid gland (it turns out that one component of therapy “interferes” with the other) [31-35]. However, in our opinion, this phenomenon is unlikely to have significant clinical significance, as evidenced by both experimental studies that studied the intrathyroidal iodine content against the background of various treatment options and clinical studies that showed high and long-term the effectiveness of fixed combinations of L-T4 and iodide, which are very popular in many countries. Here I would like to note that according to many clinical

studies that compared the effectiveness of therapy with iodide, L-T4 and their combination, the final decrease in thyroid gland (as a percentage of the initial volume) did not differ significantly between all three regimens. As already indicated, the prognosis was different: with the end of L-T4 monotherapy, goiter relapse very often developed [2, 5]. Thus, the treatment of euthyroid goiter can be presented in several stages. Initially, most patients should be treated with iodine monotherapy or combination therapy with iodine and L-T4. The latter, along with this, can be prescribed if monotherapy is ineffective. With persistent normalization of thyroid volume, an attempt should be made to return to monotherapy with iodine preparations.

Conclusion. If the volume of the remaining thyroid lobe or the total volume of the thyroid remnant is sufficient to maintain euthyroidism (more than 8-10 ml), most patients are prescribed preventive monotherapy with iodide at a dose of 200 mcg/day ("Iodine Balance-200"). If, against the background of iodide monotherapy, an increase in TSH levels is detected over time over time (subclinical hypothyroidism develops), treatment is supplemented with L-T4[35-37]. If the volume of thyroid tissue left after surgery does not allow maintaining a euthyroid state, it is most advisable for the patient to be prescribed combination therapy with L-T4 and iodine: L-T4 in this case will replace the deficiency of thyroid hormones, and iodine will help prevent recurrence of nodular formation in the left thyroid tissue. For this purpose, it is convenient to use fixed combinations containing iodide and L-T4 (Iodotirox) in one tablet.

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