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MODERN CLINICAL AND MORPHOLOGICAL ASPECTS OF SUBCLINICAL HYPOTHYROIDISM IN PREGNANCY

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Abstract:

Hypothyroidism is a reduced function of the thyroid gland. Subclinical hypothyroidism is characterized by the absence of symptoms, but at the same time, tests signal an increase in TSH (thyroid-stimulating hormone of the pituitary gland - it is responsible for regulating the secretion of thyroid hormones). Therefore, it is very important to take all the tests prescribed by the doctor on time, because timely and correct treatment can prevent the consequences of hypothyroidism.

Keywords: manifest hypothyroidism, levothyroxine, nodular goiter, thyroid peroxidase, suppression of the immune system.

Introduction

Subclinical hypothyroidism (FGT) during pregnancy is defined as exceeding the permissible values of thyroid-stimulating hormone (TSH) during pregnancy with a normal level of thyroid hormones (TG) [1, 2]. SGT is associated with health risks
mothers and children, mainly with premature birth and spontaneous abortions [2–4]. Markers of the autoimmune process in the thyroid gland are present in 2–17% of pregnant women [2, 4] and, both in isolation and together with changes in thyroid status, are associated with complications of the onset and course of pregnancy, such as spontaneous abortions.
you [5–8], miscarriage [9] and premature birth [8, 10–12]. The occurrence and indications for therapy of thyroid pathology during pregnancy depend on the studied population, gestational age, the presence of markers of the autoimmune process in the thyroid gland, as well as on the accepted reference values for TSH and free thyroxine (free T4), in connection with which the fixed reference values
TSH and free T4 levels during pregnancy are currently questioned [2, 13]. At present, there is no unified approach to the diagnosis and treatment of SGT in Russia and in the world, and therefore it is of great importance that doctors who encounter the problems of SGT in pregnant women are aware of modern ideas about approaches to this problem. Determination of the correct indications for the treatment of SHT during pregnancy is necessary, on the one hand, to prevent pregnancy complications, on the other hand, to prevent unjustified drug therapy,

psychological and financial burden on pregnant women and the health care system.

Pregnancy involves physiological changes in the level of TSH and thyroid hormones. Firstly, the similar structure of TSH and human chorionic gonadotropin (hCG) contributes to the stimulation of TSH receptors in the thyroid gland and an increase in the synthesis of thyroid hormones, as well as, according to the principle of feedback, a decrease in TSH levels in the first trimester of pregnancy. Secondly, under the influence of estrogens in the liver, the synthesis of thyroxine-binding globulin, which has a high

affinity for thyroxine. As a result, from the 7th week of pregnancy, the level of total T4 in the blood of a pregnant woman increases. Third, the work of type 3 deiodinase in the placenta increases the breakdown of thyroxine and triiodothyronine, which also requires an increase in the synthesis of thyroid hormones during pregnancy [14–16]. Finally, an increase in the filtration function of the kidneys leads to an increase in urinary iodine excretion during pregnancy and requires a higher intake of iodine in pregnancy, including to meet the increased need for thyroid hormones [14–16]. In addition, the determination of free T4 by automatic immunochemical analyzers is difficult during pregnancy due to an increase in the level of thyroxine-binding globulin and a decrease in the concentration of albumin

in the blood [2]. In developed countries, autoimmune thyroiditis is considered to be the main cause of SHT [16], and in all women with TSH levels above 2.5 mU/L, the level of antibodies to thyroid peroxidase (ATcTPO) should be determined [2, 13]. However, it is worth taking into account the suppression of the immune system during pregnancy, and therefore the titer of thyroid antibodies decreases by an average of 60% in the second half of pregnancy [17]. In a study by Korevaar T.M. et al., it was shown that women with ATcTPO levels greater than the 92nd percentile had higher TSH levels and a greater risk of TSH increases >2.5 mU/L. It was also noted that women with elevated levels of ATcTPO respond worse to hCG stimulation during pregnancy. The higher the level of ATcTPO, the worse the response in the form of changes in the level of free T4 and TSH to hCG stimulation [12]. The ability of the thyroid gland to adapt to the increased needs in its work during pregnancy is influenced by the level of iodine intake, body mass index (BMI), ethnicity, hCG level and other placental factors. Manifest hypothyroidism negatively affects the reproductive function of a woman. For example, at a TSH level of >15 mU/L, menstrual irregularities occurred in 68% compared to 12% in euthyroid women [33]. The effect of SGT on a woman's reproductive capacity is not so obvious. According to some data, an increase in TSH is observed more often among women who have difficulty conceiving [34, 35]. According to Abalovich M. et al., SGT was more common among women.

infertility compared to the control group (13.9% and 3.9%, respectively) [35]. According to the results of a retrospective study by Feldthusen A.D. et al., an increase in TSH and ATcTPO levels is associated with a decrease in fertility [36]. According to Poppe K. et al., on the contrary, an increase in TSH >4.2 mU/L among women with infertility is not more common than in the group

control [37]. In a prospective cohort study by Powden T.C. et al., which included more than 1000 women, TSH levels of >2.5 mU/L were not associated with pregnancy rates and the risk

of spontaneous abortions, including in women with elevated ABcTPO levels [38]. Thus, according to most experts, there is insufficient data in favor of the negative effect of SHT on the possibility of pregnancy [2, 39]. Indications for levothyroxine therapy in pregnancy planning and its effectiveness are also debatable at the moment. According to ATA, as of 2017, there is not enough data available,

to recommend levothyroxine therapy to women with normal TSH levels and elevated ABcTPO levels when planning pregnancy.

The question of starting treatment for subclinical hypothyroidism after a single study of TSH and the level of free T4 remains open. It is mandatory to prescribe levothyroxine (Eutirox) to pregnant women with subclinical hypothyroidism. All the above studies indicate the need for more frequent examination of the level of serum TSH in the presence of hyperlipidemia in older women and in persons with subdepressive states. Timely prescribed therapy prevents the progression of hypothyroidism symptoms. The administration of levothyroxine (Eutirox) to patients with subclinical hypothyroidism at a young age does not cause concern, and the dose can be calculated in the same way as in overt hypothyroidism, 1.6-1.8 μg/kg of body weight. The individual dose is selected under TSH control. For patients for whom the administration of levothyroxine may be associated with the risk of developing undesirable side effects, treatment is recommended to start with small doses (12.5 μg), and dose titration is carried out under TSH control once a month.

Eutirox is available in tablets in six different dosages: 25, 50, 75, 100, 125 and 150 mcg in one tablet. Such a wide range of dosages for the treatment of hypothyroidism and other thyroid diseases allows for a flexible selection and high accuracy of the dose of levothyroxine, better compensation for thyroid function and long-term adequate treatment.

The absence of the need to crush tablets increases the quality of treatment, patient adherence to the doctor's recommendations (compliance), which is extremely important to achieve adequate compensation for hypothyroidism.

If there are nodular formations in the thyroid gland, the endocrinologist can make a choice:

- conservative treatment;
- surgical treatment;
- dynamic observation.

Conservative treatment is based on the use of L-T4 in doses that have a suppressive effect on TSH. The role of TSH is to stimulate the functional activity of the thyroid gland, in addition, this effect extends to the growth of nodular formations, if any. To date, there is no clear evidence of the effectiveness of this method of treatment, as well as there is no data on what level of TSH is protective in relation to the growth of existing nodular formations or recurrence of postoperative nodular goiter. Doctors all over the world find it difficult to choose tactics for the treatment of nodular goiter. In 2004, clinical guidelines were presented by the Russian Association of Endocrinologists (RAE) for the diagnosis and treatment of nodular goiter. The final decision on treatment tactics is made by the attending endocrinologist. If a decision is made to prescribe suppressive therapy, the following restrictions should be kept in mind (RAE Consensus on Nodular Goiter, 2004):

• the advisability of prescribing such doses of levothyroxine that do not lead to the suppression of TSH production is questionable;

• Suppressive therapy is unsafe for at least certain groups of patients in terms of the development of osteopenia and cardiomyopathy.

The administration of levothyroxine (Eutirox) to patients with autoimmune thyroiditis and euthyroidism is a reasonable treatment in terms of preventing an increase in the size of the thyroid gland. But early administration of levothyroxine (Eutirox) for autoimmune thyroiditis can slow down not only the progression of the disease itself, but also through its autoimmune modulation. It can also affect the course of other autoimmune diseases. Questions remain regarding the dose of the drug, the duration of therapy, and the criteria for monitoring the effectiveness of treatment.

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