MODERN VIEWS OF THYROID DISEASES IN PREGNANCY

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Summary: The article provides a review of the literature on the characteristics of pregnancy in women with various types of diseases and endocrine disorders of the thyroid gland, risk factors for complications from both the mother and the fetus, principles of treatment depending on the type of pathology of thyroid function and gestational age. The article provides a review of the literature on modern concepts of thyroid diseases during pregnancy.

Key words: pregnancy, thyroid gland, hypothyroidism, woman, fetus.

Relevance. Diseases of the thyroid gland (TG) are the most common endocrine pathology, and their prevalence is 10–17 times more common in women than in men. The thyroid gland is a butterflyshaped gland weighing about 20 g and is located on the front surface of the neck in the lower third [1]. The function of the thyroid gland is under the control of the hypothalamic-pituitary system. Thyrotropin-releasing hormone (TRH) is synthesized in the hypothalamus. TRH, entering the pituitary gland, stimulates the synthesis of thyroid-stimulating hormone (TSH), which stimulates the activity of the thyroid gland. The thyroid gland produces two hormones: thyroxine (T4) and triiodothyronine (T3). The activity of triiodothyronine is 3–5 times higher than the activity of thyroxine, despite the fact that the thyroid gland secretes 10–20 times more thyroxine than triiodothyronine. In the blood, most of the thyroid hormones are bound to the carrier protein thyroglobulin and are inactive, while only a small percentage of the free fraction of hormones is active [2, 3, 8]. The role of thyroid hormones is multifaceted. They participate in almost all processes occurring in the human body. Thyroid hormones influence protein synthesis and cell growth, stimulate RNA synthesis in the nucleus, activate tissue respiration, play a major role in the process of growth and differentiation of tissues, affecting all types of metabolism, enhance metabolic processes, increase the need for various enzymes and, accordingly , in vitamins necessary for their synthesis [3]. Thyroid hormones affect the gonads by inhibiting the production of follicle-stimulating hormone and, conversely, increasing the release of luteinizing hormone from the pituitary gland. In addition, thyroid hormones increase the sensitivity of the ovaries to gonadotropic hormones and the endometrium to estrogens. During puberty, they actively influence the body,

stimulating, together with sex steroids, the final completion of physical, sexual and mental differentiation and promoting the formation of a normal two-phase cycle in the female body [12, 13, 14, 15]. During pregnancy, the function of the thyroid gland changes. Due to an increase in the concentration of thyroxine-binding globulins, caused by a high level of estrogens, the content of bound forms of thyroid hormones in the blood increases: total thyroxine and total triiodothyronine, but the free, active fractions of hormones do not increase. However, as outside pregnancy, it is these free fractions that provide all the metabolic and biological activity of thyroid hormones. The absence of elevated levels of total thyroxine and thyroxine-binding globulins is a prognostic sign of termination of pregnancy. Until 15 weeks of pregnancy, the fetus's needs for thyroid hormones are met by the mother's thyroid gland. In the first trimester, the formation and development of the nervous system occurs, which requires thyroid hormones. Thus, the state of the child's nervous system depends on the function of the thyroid gland of the mother with whom she entered into pregnancy, that is, it is necessary to check and correct the function of the thyroid gland at the pregnancy planning stage, and if not in time, in the very first weeks [11, 12]. To provide the mother and fetus with sufficient levels of thyroid hormones, the thyroid gland is stimulated. The main stimulator of its function is the pregnancy hormone (β-hCG). As a result, there is an increase in the level of the hormones T4 and T3 (the total level is always increased, the level of free T4 and T3 sometimes) and, accordingly, a decrease in TSH. These changes are especially pronounced in the first trimester of pregnancy with the maximum level of β-hCG. This condition is called gestational hyperthyroidism. It leads to a change in generally accepted norms in hormonal tests. In multiple pregnancies, due to high levels of β-hCG, TSH may drop to zero [19, 18]. Taking large doses of iodine (a favorite folk method: a few drops of iodine in milk) does not lead to termination of pregnancy, but it damages the function of the fetal thyroid gland [15]. In addition, the removal of iodine from the mother's body increases and her need for it increases. In non-pregnant women, the iodine requirement is 150 mcg, in pregnant women - 200 mcg per day [13, 16, 19]. Hypothyroidism is a condition caused by decreased function of the thyroid gland and is characterized by a reduced level of thyroid hormones in the body. The condition, regardless of the specific cause that caused the decrease in the functional activity of the gland, is usually called primary hypothyroidism. Severe forms of hypothyroidism are called myxedema; with athyroidism, the phenomena of cretinism develop. Hypothyroidism in women of reproductive age, including pregnant women, is detected in 2– 3% of cases. In 90–95% of cases this is primary hypothyroidism. A decrease in the level of thyroid hormones, which affect the physiological functions and metabolic processes in the body, leads to inhibition of all types of metabolism, oxygen utilization by tissues, and a decrease in the activity of various enzyme systems, gas exchange and basal metabolism. Slowing

down the synthesis and catabolism of protein, as well as its excretion from the body, leads to a significant increase in the amount of protein breakdown products in organs and tissues, skin and muscles [8, 10]. Women with hypothyroidism rarely become pregnant. Reproductive function in women suffering from hypothyroidism is sharply depressed. A significant slowdown in metabolic and trophic processes affects the function of the ovaries: there is a delay in the maturation of primordial follicles, ovulation and the development of the corpus luteum are disrupted. Untreated or uncompensated hypothyroidism in women affects conception and increases the incidence of spontaneous abortions and stillbirths. Impaired conception may be a consequence of secondary hyperprolactinemia, causing anovulatory menstrual cycles. Uncompensated hypothyroidism leads to infertility, premature termination of pregnancy, and in the case of full-term pregnancy, defective children are born [11, 13, 14]. Clinical signs and symptoms of hypothyroidism in pregnant women. Because thyroid hormone receptors are present in virtually all tissues, the symptoms of hypothyroidism are many and varied. Their severity depends on the degree and duration of thyroid hormone deficiency. The most typical clinical signs of hypothyroidism are: general weakness, lethargy, drowsiness, memory loss, dry skin, hair loss, brittle nails, constipation, chilliness, muscle pain, bleeding gums, tooth decay, pale skin, swelling, slow speech, sluggishness movements, hoarse voice, heart rate 52–60 beats. per minute against the background of a decrease in TSH levels with low or normal levels of thyroid hormones [8, 14, 17]. As the duration of pregnancy increases, the symptoms of hypothyroidism in patients constantly taking medications decrease: in the second half of pregnancy, symptoms of hyperfunction of the thyroid gland appear, primarily tachycardia. This is a consequence of a compensatory increase in the function of the fetal thyroid gland and the flow of thyroid hormones from the fetus to the mother [17, 23, 22]. Typical complications in pregnant women with hypothyroidism are: eclampsia, intrauterine fetal death, profound developmental anomalies in newborns, miscarriage, premature birth, persistent weakness of labor, and the development of iron and folate deficiency anemia. According to modern concepts, the vast majority of women with thyroid pathology, after appropriate treatment under the supervision of an endocrinologist, can plan a pregnancy, which, with adequate control, will not be associated with a significant increase in the risk of obstetric and gynecological complications and the development of pathology in the fetus [1, 5]. Pregnancy without delay (in the absence of other contraindications) can be planned in women: with compensated primary hypothyroidism that developed as a result of AIT or surgical treatment of non-tumor thyroid diseases; various forms of euthyroid goiter (nodular, multinodular, mixed), when there are no direct indications for surgical treatment (significant size of nodular goiter, compression syndrome); identified carriage of antibodies to the thyroid gland in the absence of impairment of its function.

During pregnancy, all women belonging to the listed groups require a dynamic assessment of thyroid function, which involves determining the level of TSH and thyroid hormone. T4 in every trimester of pregnancy. In addition, women with goiter should undergo dynamic ultrasound [8]. Women with uncompensated hypothyroidism as a result of AIT or after surgical treatment of non-tumor thyroid pathology can plan pregnancy after achieving euthyroidism against the background of L-thyroxine replacement therapy. Patients with thyrotoxicosis, after achieving stable remission, can plan pregnancy after two years. If radioactive iodine therapy was carried out, pregnancy should be postponed for one year [22]. It is fundamentally important to have trained specialists in this field of endocrinology and to adhere to programs for screening, diagnosing and treating thyroid dysfunction in pregnant women.

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