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HYPOTHYROIDISM AND REPRODUCTIVE DYSFUNCTION IN WOMEN

Komiljonova Oygul Olimjonovna
Assistant of the Department of Clinical Sciences
Asian International University, Bukhara, Uzbekistan
E-Mail: komiljonovaoygulolimjonovna@oxu.uz

Annotation: The female reproductive system is a set of interconnected structural elements: the hypothalamus, pituitary gland, ovaries, target organs and other endocrine glands that ensure the implementation of the generative function. The thyroid gland (TG) is the most important part of the neuroendocrine system, which has a significant impact on reproductive function. Thyroid hormones are necessary for the normal functioning of almost all organs and systems of the body. The problem of thyroid dysfunction in women suffering from infertility has been of great interest to scientists and clinicians in recent years. Thus, the prevalence of thyroid function disorders identified during examination of women attending infertility diagnostic and treatment clinics ranges from 2.48 to 38.3%. Hypothyroidism predominates in the structure of thyroid disorders in infertile women, while its frequency varies significantly, reaching, according to some data, 78.4%. According to the results of recent studies, hypothyroidism (both manifest and subclinical) is a fairly significant factor in female infertility.

Key words: Hypothyroidism, reproductive function, Thyroid hormones, thyroid-stimulating hormone – TSH.

The problem of restoring reproductive function does not lose its relevance, since the frequency of infertile marriages in recent decades remains high (10–15%) and does not tend to decrease. The female reproductive system is a set of interconnected structural elements: the hypothalamus, pituitary gland, ovaries, target organs and other endocrine glands that ensure the implementation of the generative function. The thyroid gland (TG) is the most important link in the neuroendocrine system, which has a significant impact on reproductive function. The main function of the thyroid gland is to provide the body with thyroid hormones: thyroxine (T4) and triiodothyronine (T3), an integral structural component of which is iodine. Thyroid hormones are necessary for the normal functioning of almost all organs and systems of the body. They regulate the processes of development, maturation, specialization and renewal of almost all tissues and are of exceptional importance for the formation and development of the fetal brain, the formation of the child's intelligence, the growth and maturation of the bone skeleton, the reproductive system, and influence sexual development, menstrual function and fertility.

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The close connection between the hypothalamic-pituitary-ovarian and hypothalamic-pituitary-thyroid systems is due to the presence of common central regulatory mechanisms. The function of the reproductive and thyroid systems is regulated by tropic hormones of the anterior pituitary gland (luteinizing hormone - LH, follicle-stimulating hormone - FSH, prolactin - PRL, thyroid-stimulating hormone - TSH), which in turn are under the control of the hypothalamus. Thyrotropin-releasing hormone (TRH, thyroliberin) of the hypothalamus is a stimulator of not only TSH, but also PRL of the pituitary gland, therefore dysfunction of the pituitary-thyroid system leads to changes not only in gonadotropins, but also in PRL. Regulation of the synthesis and secretion of thyroid hormones, on the one hand, is carried out by the effects of TSH, and on the other, by autoregulatory processes occurring in the thyroid gland itself, which depend on the level of iodine consumption and the synthesis of thyroid hormones. The release of TSH is regulated by the hypothalamic tripeptide TRH and the level of free thyroid hormones. The latter type of regulation is carried out due to the effects of T3 on specific nuclear receptors in thyrotrophs. In addition, the level of thyroid hormones influences the hypothalamic production of TRH. TRH from the hypothalamus stimulates the production of TSH in the pituitary gland; TSH stimulates the production of thyroid hormones by the thyroid gland; the latter, through a negative feedback mechanism, suppress the production of TSH and TRH. Having reached thyrocytes, TSH interacts with receptors located on the cell membrane. The binding of TSH to the receptor leads to the activation of adenylate cyclase and a number of other post-receptor mechanisms. As a result, various functions of the thyrocyte are stimulated, in particular the uptake of iodine and its active transport through the basement membrane, the synthesis of thyroglobulin and the release of thyroid hormones.

It has been established that LH, FSH and TSH are complex glycoproteins consisting of a- and b-subunits. The structure of the a-subunit of LH, FSH and TSH is the same, and the b-subunit is specific for each hormone and determines its luteinizing, follicle-stimulating and thyroid-stimulating activity only after combining with the a-subunit. The discovered structural similarity allowed us to conclude that these hormones originated from one precursor during the process of evolution and that changes in the content of some hormones could influence others. Thyroid pathology can cause premature or late puberty, menstrual irregularities, infertility, galactorrhea, miscarriage, pathology of the fetus and newborn. In turn, the state of the reproductive system has a significant impact on thyroid function. This is confirmed by changes in thyroid function during pregnancy and lactation in patients with benign tumors and hyperplastic processes of the female genital organs . It has now been proven that estrogens have a pronounced stimulating effect on the thyroid gland, primarily due to

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the intensification of the synthesis of thyroxine-binding globulin (TBG) in the liver. In addition, estrogens increase the sensitivity of pituitary thyrotrophs to thyroliberin.

Pregnancy is accompanied by the influence of a complex of factors specific to this condition, which together lead to significant stimulation of the pregnant woman's thyroid gland. Such specific factors are: hyperproduction of human chorionic gonadotropin (HCG); increased production of estrogen and thyroxine-binding globulin (TBG); an increase in renal blood flow and glomerular filtration, leading to increased excretion of iodine in the urine; changes in the metabolism of maternal thyroid hormones due to the active functioning of the fetoplacental complex. For almost the entire first half of pregnancy, the fetal thyroid gland is not yet functioning and its development is fully dependent on the thyroid hormones of the pregnant woman. Therefore, the need for thyroid hormones during pregnancy increases by 40–50%, and hypothyroxinemia of any origin has the most adverse consequences precisely in the early stages of pregnancy.

The problem of thyroid dysfunction in women suffering from infertility has attracted great interest among scientists and clinicians in recent years. Thus, the prevalence of thyroid function disorders identified during examination of women attending infertility diagnostic and treatment clinics ranges from 2.48 to 38.3% . Hypothyroidism predominates in the structure of thyroid disorders in infertile women, while its frequency varies significantly, reaching, according to some data, 78.4% , which is apparently due to the heterogeneity of samples and the specificity of the clinics to which infertile couples turn . According to the results of recent studies, hypothyroidism (both manifest and subclinical) is a fairly significant factor in female infertility.

From the point of view of etiology, hypothyroidism is divided into primary (thyroidogenic), secondary (pituitary), tertiary (hypothalamic) and tissue (transport, peripheral). The vast majority of cases of hypothyroidism are caused by thyroid pathology (primary hypothyroidism). Most often, primary hypothyroidism develops as a result of autoimmune thyroiditis (AIT), less often after thyroid surgery and radioactive iodine therapy. Secondary and tertiary hypothyroidism, which develops as a result of TSH and TRH deficiency, respectively, are rarely observed; their differential diagnosis in clinical practice presents significant difficulties, and therefore they are often combined with the term central (hypothalamic-pituitary) hypothyroidism. Primary hypothyroidism, which developed as a result of destruction of the thyroid gland itself due to AIT, has the greatest clinical significance and distribution in women of reproductive age.

The classification of primary hypothyroidism by severity is based primarily on laboratory diagnostic data taking into account clinical manifestations:

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1. Subclinical - the concentration of TSH in the blood is increased, free T4 is within normal limits; usually asymptomatic or only nonspecific symptoms.

2. Manifest – the concentration of TSH in the blood is increased, free T4 is decreased; characteristic symptoms of hypothyroidism (usually also nonspecific) are usually present, but an asymptomatic course is also possible. A. Compensated. B. Decompensated.

3. Complicated - a detailed clinical picture of hypothyroidism, severe complications: polyserositis, heart failure, cretinism, myxedematous coma, etc. It should be remembered that the classic clinical manifestations of manifest hypothyroidism ("mask-like" face, swollen limbs, obesity, decreased body temperature, slow speech , hoarse voice, drowsiness, lethargy, paresthesia, memory loss, thinning of scalp hair, hyperkeratosis of the skin of the elbows, anemia, biliary dyskinesia, depression, etc.) are very diverse, nonspecific and never occur at the same time.

The TSH level is one of the indicators for predicting the effectiveness of assisted reproductive technology (ART) programs and indicates the important role of thyroid hormones in oocyte physiology. A. Geva et al. found that AT-TG were present in 20% of women with infertility who required IVF and ET for tubo-peritoneal factor infertility and infertility of unknown origin, while antiovarian antibodies were detected in 12%. It is noteworthy that all women in this study had no thyroid dysfunction at the time of the examination or in the anamnesis. These results suggest that antithyroid antibodies may be an independent factor in infertility.

There are other points of view on this problem. Some researchers deny the relationship between elevated levels of AT-thyroid and spontaneous miscarriages in women of reproductive age and believe that the presence of AT-thyroid, detected before pregnancy, does not increase the risk of miscarriage in women without a history of miscarriage and does not affect the possibility of pregnancy in these women. Currently, there is no single point of view on the role of AT-thyroid gland in the pathogenesis of infertility and miscarriage. The following hypotheses are discussed in the literature. The first hypothesis suggests that women with elevated AT-thyroid levels develop subclinical hypothyroidism, which contributes to decreased fertility or leads to spontaneous miscarriage in early pregnancy. The second hypothesis considers AT-TG as markers of predisposition to autoimmune diseases, and not the direct cause of miscarriage. As a third hypothesis, it is suggested that AT-TG serve as peripheral markers of dysfunction of T-lymphocytes. The fourth hypothesis is that autoimmune thyropathies for one reason or another lead to the fact that in women with AT-thyroid gland pregnancy occurs at an older age, which in itself increases the risk of miscarriage. Despite the large number of hypotheses about the relationship between carriage of antithyroid antibodies and miscarriage, unambiguous data on this issue have not yet

been obtained. Nevertheless, all authors are unanimous in the opinion that the risk of spontaneous abortion in the early stages in women with AT-TG exceeds that in women without them by 2–4 times, therefore, carriers of AT-TPO constitute a risk group for early reproductive losses, which requires special observation of this category of women by obstetricians and gynecologists even at the stage of pregnancy planning. The introduction and rapid development of ART in recent years has led to a significant increase in the proportion of induced pregnancies, and the problem of maintaining these pregnancies and the birth of healthy children has become particularly urgent. Induced pregnancy (IP) is a pregnancy that occurs as a result of the use of ovulation inducers - drug stimulants of ovulation, widely used to restore fertility in anovulatory forms of infertility and in IVF and PE programs. Induction of ovulation is accompanied by the simultaneous growth of several, and sometimes many, follicles, in contrast to the spontaneous cycle, and, accordingly, the formation of many corpora lutea. These hormonally active structures secrete steroid hormones, the concentration of which is tens of times higher than physiological ones.

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