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THE IMPACT OF THYROID DYSFUNCTION ON FEMALE REPRODUCTIVE FUNCTION: A CLINICAL CASE

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The impact of thyroid dysfunction (primary and secondary hypothyroidism, Graves' disease, functional autonomy) on female reproductive function is discussed. A clinical case of secondary hypothyroidism of adrenal origin affecting the development of infertility is presented.

The aim of the study. *To review the literature on the manifestations of reproductive function disorders and the assessment of thyroid status in women of reproductive age.*

Research results*. In the case of thyroid hormone deficiency, there is a decrease in the gonadotropic function of the pituitary with the development of hyperprolactinemia and luteal phase insufficiency of the menstrual cycle. A reduction in the synthesis of sex steroid-binding globulin increases the level of free T4, which can cause symptoms similar to polycystic ovary syndrome. Hypothyroidism is a common cause of female infertility. In hyperthyroidism, women experience hyperestrogenism, leading to a reduction in follicle-stimulating hormone levels. Progesterone levels remain low due to reduced ovarian tissue sensitivity to luteinizing hormone in the context of follicle-stimulating hormone deficiency. Pregnancy is possible in women with Graves' disease, but its course, in the absence of adequate treatment, is characterized by spontaneous miscarriage in 70% of patients.*

In primary hypothyroidism, the decreased production of thyroid hormones leads to increased secretion of thyroliberin, which further stimulates prolactin secretion, helping to explain the genesis of galactorrhea. It also reduces progesterone secretion by the ovaries. This results in the development of opsomenorrhea, amenorrhea, and infertility.

One of the causes of central hypothyroidism may be adrenal-origin hyperandrogenism. Since hyperandrogenism blocks TSH secretion, the level of thyroid hormones in the blood decreases. Thyroliberin is activated, leading to the disruption of reproductive function.

Conclusions. *In cases of reproductive function disorders in women of reproductive age, it is essential to assess thyroid status and, if necessary, prescribe appropriate therapy. The issues discussed require further in-depth study, as they open up fundamentally new perspectives in the treatment of reproductive system dysfunctions in women*

Keywords: *hypothyroidism, hyperthyroidism, thyroliberin, hyperandrogenism, infertility*

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1. Introduction

It is well known that thyroid diseases (TD) accompanied by thyroid dysfunction can lead to menstrual cycle (MC) disorders, decreased fertility, pregnancy loss, and fetal development pathology. Pathological processes in the thyroid gland that are associated with changes in the production of thyroid hormones (T3 and T4) affect the hormonal profile parameters in women, altering the balance of neuroendocrine mediators and resulting in the development of reproductive dysfunction [1, 2]. Antibodies to the thyroid gland are present in 5–10% of women, which may be one of the causes of infertility. Antithyroid antibodies may also be markers of generalized autoimmune dysfunction in the body, ultimately leading to pregnancy loss.

In regions with moderate iodine deficiency, thyroid pathology reflects the final stage of the pathomorphosis of iodine deficiency diseases, particularly multinodular toxic goiter as the extreme manifestation of thyroid functional autonomy [3].

In women with thyroid hormone deficiency, there is a reduction in the gonadotropic function of the pituitary gland, leading to hyperprolactinemia and luteal phase insufficiency in the menstrual cycle (MC). Due to the decreased synthesis of sex steroid-binding globulin (SSBG), the level of free T4 increases, which can cause symptoms similar to those seen in polycystic ovary syndrome (PCOS). Hypothyroidism is traditionally considered a common cause of female infertility. In hyperthyroidism, women experience hyperestrogenism, which, through a feedback mechanism, leads to a decrease in the concentration of follicle-stimulating hormone (FSH). The level of progesterone remains quite low due to the decreased sensitivity of

ovarian tissues to luteinizing hormone (LH) under conditions of FSH deficiency [3, 4].

It has been established that although pregnancy is possible in women with Graves' disease, its course, in the absence of adequate thyroid toxicosis control, is characterized by spontaneous abortion in 70% of patients. In cases where timely detection and thyrostatic therapy are applied, favorable pregnancy outcomes were observed in 73%. Therefore, it is advisable to include the assessment of thyroid function in the diagnostic algorithm for pregnant women and those with menstrual cycle disorders, infertility, and pregnancy loss.

There is a well-supported opinion that thyroliberin stimulates the synthesis of prolactin. In primary hypothyroidism, a decrease in thyroid hormone production leads to increased secretion of thyroliberin by the hypothalamus, which, in addition to influencing TSH production, also stimulates prolactin secretion. This mechanism helps explain the genesis of galactorrhea. Elevated prolactin levels in the blood reduce progesterone secretion by the ovaries or, by disrupting the cyclic secretion of gonadotropins, interfere with their effect on the ovaries. This results in menstrual cycle (MC) disorders, the development of oligo- and amenorrhea, and infertility. This pathology is known in the literature as Van Wyk–Ross Hennes syndrome [5].

One of the rare causes of central (secondary) hypothyroidism may be hyperandrogenism (in our case, adrenal genesis). Since hyperandrogenism blocks TSH secretion [6–8], the level of thyroid hormones in the blood decreases accordingly with lower TSH levels. Under these conditions, thyroliberin is activated, which, as mentioned earlier, leads to menstrual cycle (MC) disturbances and reproductive dysfunction. This case is presented in more detail. It is also important to consider the general therapeutic, neurological, and gynecological masks of hypothyroidism [9, 10].

2. Clinical case description

Patient N., 29 years old, married for 5 years. She complained of infertility, menstrual cycle (MC) disturbances, drowsiness, decreased work capacity, memory problems, and excessive hair growth.

Objective examination. Height: 165 cm, weight: 70 kg, feminine body type. Skin slightly pigmented, hirsutism grade II according to the Ferriman-Gallwey scale. The thyroid gland is not palpable. Heart rhythm is regular, pulse 72/min, blood pressure 130/80 mm Hg. Vesicular breathing. The liver extends 1 cm below the costal margin.

Gynecological consultation. The breasts are infantile, no masses detected. When pressing on the areolas, a drop of colostrum is released from the nipples. The clitoris is hypertrophied, the labia minora are hypoplastic. The uterus and appendages are unremarkable. Basal body temperature shows an elongated follicular phase and shortened luteal phase of the cycle.

Laboratory and instrumental examination data:

– Blood Hormones:

TSH: 0.15 mU/ml (normal 0.4–4.2)

Free T4: 0.35 ng/dl (normal 0.70–1.48)

Prolactin: 32.1 ng/ml (normal 2.58–18.12)

Free testosterone: 12.3 ng/dl (normal < 2.0)

Dehydroepiandrosterone sulfate (DHEA-S): 746 µg/dl (normal 95.8–511.7)

17-Hydroxyprogesterone: 103.9 ng/ml (normal < 2.0)

– Ultrasound: Thyroid gland of normal size and structure. Ovaries: volume up to 6 cm^3, follicles at various stages of maturity. Uterus underdeveloped

– CT: Adrenal glands of normal shape, moderate bilateral hyperplasia.

Diagnosis. Classic form of adrenal genital syndrome. Secondary hypothyroidism, moderate severity. Primary infertility. The diagnosis was verified by the aforementioned results of laboratory and instrumental examinations.

Treatment. Levothyroxine 75 mcg/day for hypothyroidism. Prednisolone 5 mg twice a day to correct adrenal gland hormone function. Diane-35 (24/7 regimen) to reduce hirsutism. After 2.5 months of treatment, galactorrhea decreased, memory improved, and there was a trend toward normalization of the MC. Ongoing monitoring of the patient continues.

3. Conclusions

1. In the presented clinical case of reproductive dysfunction in a patient of fertile age, along with determining androgen levels in the blood, thyroid status should be evaluated, and appropriate therapy prescribed if necessary.

2. The issues discussed require further in-depth study, as they open up fundamentally new perspectives in the treatment of reproductive system dysfunctions in the female body.

Conflict of interest

The authors declare that they have no conflict of interest regarding this study, including financial, personal, authorship, or any other type of conflict that could influence the research and its results presented in this article.

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Data availability

Data will be made available on reasonable request.

Use of artificial intelligence

The authors confirm that they did not use artificial intelligence technologies in creating the submitted work.

Authors' contributions

Ruslana Liashuk: Conceptualization, Data Curation, Formal Analysis, Investigation, Methodology, Project Administration, Resources, Software, Supervision, Validation, Visualization, Original Draft, and Writing.

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