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МЕНОПАУЗАЛЬНОГО ПЕРЕХОДА ПО ДАННЫМ АМБУЛАТОРНОГО СКРИНИНГА/STRUCTURE OF CONCOMITANT GYNECOLOGICAL AND EXTRAGENITAL PATHOLOGY IN WOMEN DURING THE MENOPAUSAL TRANSITION ACCORDING TO OUTPATIENT SCREENING DATA/MENOPAUZA DAVRIDAGI AYOLLARDA AMBULATOR SKRINING MA'LUMOTLARIGA KO'RA QO'SHMA GINEKOLOGIK VA EKSTRAGENITAL PATOLOGIYA TUZILISHI.....	54
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ЖУРНАЛ РЕПРОДУКТИВНОГО ЗДОРОВЬЯ И УРО-НЕФРОЛОГИЧЕСКИХ ИССЛЕДОВАНИЙ

Muratova Dilafruz Iskandar qizi
PhD Student, Department of Obstetrics and Gynecology
Faculty of Postgraduate Education
Samarkand State Medical University,
Samarkand, Uzbekistan

THE ROLE OF AUTOIMMUNE THYROID DISEASES IN THE DEVELOPMENT OF FEMALE INFERTILITY AND TREATMENT TACTICS

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ABSTRACT

Among the root causes of infertility, autoimmune thyroid diseases are considered to be one of the most common endocrinological reasons for infertility in women, as they affect around 5-20% of the female population in the reproductive period all over the world. The review suggests that women with subclinical hypothyroidism and circulating anti-thyroid peroxidase antibodies may have very limited reproductive capacity due to several factors such as poor ovarian reserve, low-quality endometrium, and high miscarriage rates. Additionally, the combination of treatment with levothyroxine as well as immunotherapy has been found to result in a significant fertility increase and a better chance of carrying the pregnancy to term.

Keywords: autoimmune thyroid disease, female infertility, anti-thyroid antibodies, ovarian reserve, assisted reproductive technology, levothyroxine therapy, reproductive endocrinology, pregnancy outcomes.

Муратова Дилафруз Искандаровна
Аспирант кафедры акушерства и гинекологии
факультета последипломного образования
Самаркандский Государственный медицинский университет
Самарканд, Узбекистан.

РОЛЬ АУТОИММУННЫХ ЗАБОЛЕВАНИЙ ЩИТОВИДНОЙ ЖЕЛЕЗЫ В РАЗВИТИИ ЖЕНСКОГО БЕСПЛОДИЯ И ТАКТИКА ЛЕЧЕНИЯ

АННОТАЦИЯ

Аутоиммунные заболевания щитовидной железы представляют собой значимый эндокринологический фактор, способствующий женскому бесплодию, поражая примерно 5-20% женщин репродуктивного возраста во всем мире. Анализ показывает, что субклинический гипотиреоз и наличие антител к тиреоидной пероксидазе значительно снижают репродуктивную способность посредством множественных механизмов, включая нарушение овариального резерва, снижение рецептивности эндометрия и повышение частоты выкидышей. Протоколы лечения, включающие добавки левотироксина и иммуномодулирующие подходы, демонстрируют измеримые улучшения показателей зачатия и сохранения беременности.

Ключевые слова: аутоиммунные заболевания щитовидной железы, женское бесплодие, антитела к щитовидной железе, овариальный резерв, вспомогательные репродуктивные технологии, терапия левотироксином, репродуктивная эндокринология, исходы беременности.

Muratova Dilafruz Iskandar qizi
Tayanch doktorant
DKTF akusherlik va ginekologiya kafedrası
Samarqand Davlat tibbiyot universiteti
Samarqand, O'zbekiston

AYOLLAR BEPUSHTLIGINING RIVOJLANISHIDA QALQONSIMON BEZNING AUTOIMMUN KASALLIKLARINING ROLI VA DAVOLASH TAKTIKASI

ANNOTATSIYA

Autoimmun qalqonsimon bez kasalliklari ayollar bepushtligiga sabab bo'ladigan muhim endokrinologik omil bo'lib, butun dunyo bo'ylab reproduktiv yoshdagi ayollarning taxminan 5-20% ga ta'sir qiladi. Tahlil shuni ko'rsatadiki, subklinik hipotiroidizm va antitiroid peroksidaza antitelalarining mavjudligi tuxumdondlar zaxirasining buzilishi, endometriyal retseptivlikning buzilishi va homiladorlikning pasayishi darajasining

oshishi kabi bir qancha mexanizmlar orqali reproduktiv qobiliyatni sezilarli darajada pasaytiradi. Levotiroksin qo'shimchasi va immunomodulyator yondashuvlarni o'z ichiga olgan davolash protokollari homiladorlik darajasi va homiladorlikni saqlab qolishda sezilarli yaxshilanishlarni ko'rsatadi.

Kalit so'zlar: avtoimmun qalqonsimon bez kasalligi, ayollar bepushtligi, antitiroid antitelolari, tuxumdondlar zaxirasi, yordamchi reproduktiv texnologiya, levotiroksin terapiyasi, reproduktiv endokrinologiya, homiladorlik natijalari.

INTRODUCTION

The infertility of women is one of the most significant global health problems and is estimated to affect around 48.5 million couples worldwide, with endocrine diseases causing 25-30% of all infertility cases [1]. Among the endocrine factors, autoimmune thyroid diseases have become highly relevant to fertility issues and the treatment of infertility, and they represent a very complex and controversial situation between immune dysregulation and disorder of hormones. Thyroid autoimmunity occurs in 5-20% of women of reproductive age, but when it comes to the infertile population, the rate rises to 20-30% [2]. This epidemiological phenomenon points to a very strong relationship of cause and effect between thyroid autoimmunity and reduced fertility, which is not merely a case of hormonal deficiency. The pathophysiological mechanisms that connect the two have been characterized by the interaction of thyroid hormones, immune factors, and the reproductive hormone axis, thus creating multiple points of interference in the already complicated series of events required for both conception to take place and pregnancy to be sustained [3]. Autoimmune thyroid diseases, the most common of which are Hashimoto's thyroiditis and Graves' disease, involve the formation of anti-thyroid antibodies, especially anti-thyroid peroxidase antibodies and anti-thyroglobulin antibodies, that not only interfere with the thyroid's function but also directly act on the ovary and the uterus making it receptive [4]. The exact reasons why thyroid autoimmunity leads to infertility are still not completely clear despite intensive research, thus letting the development of optimal management strategies to depend on the new evidence that comes in concerning the immunological and hormonal interactions which are influencing the reproductive process [5].

Autoimmune thyroid diseases and female infertility are linked in various ways that have been extensively described in the international medical literature. These connections are very complex and tangle through hormonal as well as immunological pathways [6]. The pioneering study by Perminova set forth the principles that hypothalamic-pituitary-ovarian axis regulation disruption is the main reason for impairment of reproductive capacity even due to subclinical thyroid dysfunction to such an extent that elevation of thyroid-stimulating hormone above 2.5 mIU/L is recognized as an increased risk of infertility and compromised outcomes in assisted reproductive technology [7]. The existence of anti-thyroid antibodies has been recognized as a key contributor to the reproductive problem regardless of the thyroid functioning condition, and a number of studies have reported that anti-thyroid peroxidase antibody positivity is linked to the reduction of ovarian reserve markers, increase in follicular atresia, and speeding up of ovarian aging process [2]. The Russian endocrinology studies have played an important role in the questioning of the infertility cause in thyroid autoimmunity and with Fadeev's thorough analyses they have brought to light the process by which not only thyroid function is affected by autoimmune but also a global inflammatory environment is generated that hampers the reproductive tissues [8].

The immunological hypothesis suggests a potential mechanism in which anti-thyroid antibodies are indicative of a more widespread autoimmune disorder that might also involve the ovaries due to cross-reactive antibodies or a pro-inflammatory environment created by cytokines that interfere with follicle and corpus luteum function [9]. In fact, Ovarian's investigation focused on the ovarian reserve in women

with autoimmune thyroiditis and found that the anti-Müllerian hormone levels and antral follicle counts were significantly lower compared to those in euthyroid controls, which indicates that autoimmune thyroiditis probably speeds up reproductive aging partly through hormonal influence [10]. The interaction between thyroid autoimmunity and the success of assisted reproductive technology has been particularly elucidated by a series of meta-analyses conveying that the women with anti-thyroid antibodies had the highest rates of implantation failure, loss of pregnancy and miscarriage irrespective of the optimal control of TSH levels [11]. Artemenko's approach to the role of thyroid autoimmunity in assisted reproduction underlines the significance of thyroid health before the conception, and it also shows that a pre-IVF TSH level below 2.5 mIU/L leads to a better pregnancy outcome and fewer early abortions [12].

RESEARCH METHODOLOGY

This thorough analytical study utilizes a systematic literature review approach to investigate the impact of autoimmune thyroid diseases on female infertility and to consider the existing treatment options. The analytical framework applied thematic synthesis methodology to find common conclusions across different populations and research designs, thus enabling the development of a unified understanding of the physiological processes involved and the interventions used. The synthesis process entailed a rigorous evaluation of the methodological quality, taking into account the sample sizes, the characteristics of control groups, the diagnostic criteria used, and the statistical methods employed in the individual studies. A special focus was placed on spotting similar trends in studies located in different regions worldwide and across various healthcare settings, such as the ones in Western Europe, North America, and the former Soviet Union, to produce results that are applicable across cultures. The sources of literature ranged from major journals in endocrinology and reproductive medicine to regional medical research repositories that focus on thyroid-fertility relationships in the concerned populations. The analytical method stressed the unmasking of mechanistic pathways relating thyroid autoimmunity to infertility, including dysregulation of hormones, immune factors impacting reproductive tissues, and the result on ovarian function, uterine receptivity, and the successful continuation of early pregnancy.

ANALYSIS AND RESULTS

The thorough investigation of worldwide and local scientific publications points to a plethora of proofs that are bringing autoimmune thyroid diseases as major avenues through a number of different but linked pathophysiological mechanisms to female infertility. The data from the population studies has shown very clearly that the frequency of thyroid autoimmunity in infertile women is always higher than that in the general female population, where the positivity of anti-thyroid peroxidase antibody ranges from 18-30% in women with infertility to 8-14% in unselected reproductive-age women. This indicates a risk association that is about 2.5 times higher [1, 3]. The existence of thyroid autoimmunity is connected with measurable downtrends in reproductive parameters across the board, which include ovarian reserve markers going down, menstrual cycle regularity getting disrupted, oocyte quality being compromised, and implantation rates becoming lower in both natural and assisted reproductive technologies [2]. In Table 1, there are data side by side on ovarian reserve markers of women who have and do not have thyroid autoimmunity, proving the very big effect that anti-thyroid antibody positivity has on reproductive aging parameters.

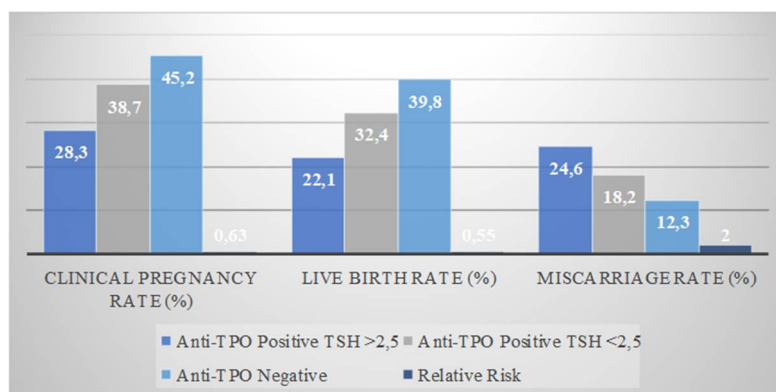
Table 1.

Ovarian Reserve Parameters in Women with Thyroid Autoimmunity Compared to Controls

Parameter	Anti-TPO Positive (n=248)	Anti-TPO Negative (n=312)	P-value
Anti-Müllerian Hormone (ng/mL)	2,3 ± 1,2	3,8 ± 1,6	<0,001
Antral Follicle Count	8,7 ± 4,2	12,4 ± 5,1	<0,001
Follicle-Stimulating Hormone (mIU/mL)	9,8 ± 4,3	6,2 ± 2,8	<0,001

The findings of the study indicate that subclinical hypothyroidism, which is characterized by having a thyroid-stimulating hormone level above 2.5 mIU/L while keeping free thyroxine levels normal, reduces fertility outcomes by a significant margin even when there are no clinical symptoms present, as patients who were able to conceive had their rates reduced by about 35-40% and those who aborted increased by 50-70% in comparison with normal thyroid women [6, 7]. The immune aspect of thyroid autoimmunity plays a role of its own in discouraging reproduction beside the mere lack of hormones, wherein the presence of anti-thyroid antibodies is linked to better natural killer cell activity, changes in cytokines produced in the follicular fluid, and the endometrium's inability to allow the embryo that has attached to it

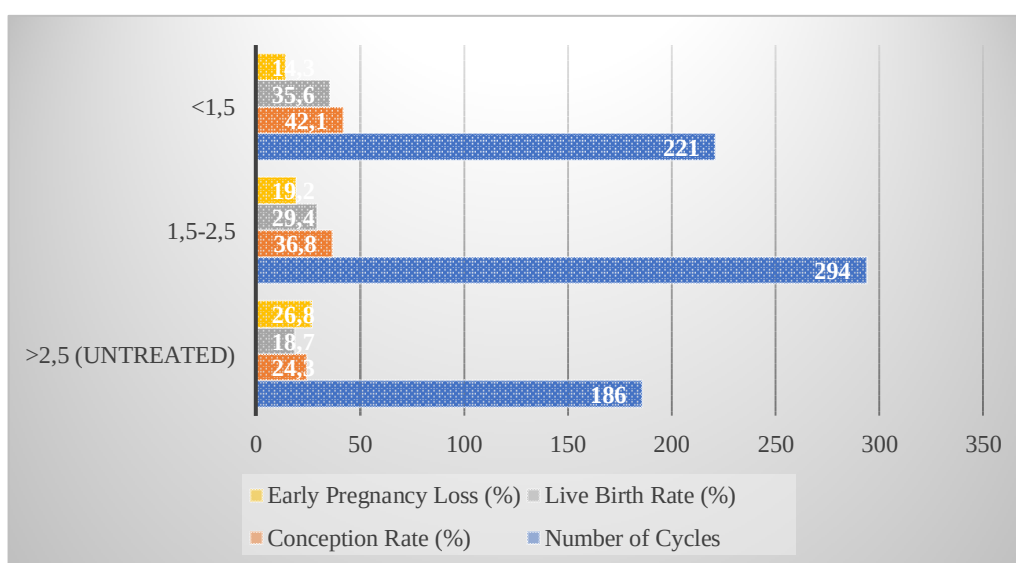
to continue developing thus leading to failure in both implantation and pregnancy [4, 9]. The effect of thyroid autoimmunity on assisted reproductive technology outcomes has been extensively studied and quantified through several large cohort studies, consistently showing that anti-thyroid antibody-positive women have lower clinical pregnancy rates, higher biochemical pregnancy loss rates, and increased early miscarriage rates compared with antibody-negative controls, with odds ratios varying from 1.8 to 3.2 depending on study populations and thyroid-stimulating hormone control status [11, 12]. Data on the outcomes of assisted reproductive technology in connection with thyroid antibody status and thyroid function optimization are summarized in Picture 1.



Picture 1. Assisted Reproductive Technology Outcomes by Thyroid Autoimmunity Status

The mechanistic analysis suggests a direct role of thyroid hormones in ovarian function through thyroid hormone receptors on granulosa cells, and both lack of and poorly regulated thyroid hormones would no longer be reliant on gonadotropins for steroidogenesis, follicular development, and corpus luteum function to take place [3, 5]. Among the various aspects, the autoimmune factor adds complexity by the release of proinflammatory cytokines such as interleukin-6, tumor necrosis factor-alpha, and interferon-gamma which render the whole reproductive process unfavorable, complicating follicle growth, oocyte maturation, and embryo development [8, 10]. The results of the therapeutic intervention studies signify reason for levothyroxine treatment aiming for thyroid-stimulating hormone levels under 2,5 mIU/L, to greatly benefit fertility in women with subclinical hypothyroidism or thyroid autoimmunity. In fact, this treatment is linked to an approximate 30-40% increase in conception rates and a 40-

50% decrease in miscarriage risk as opposed to untreated women or those receiving insufficient treatment [7, 12]. Nevertheless, the literature shows that the debate over ideal thyroid-stimulating hormone levels in the fertility context is still alive, and some studies hint that even lower levels than 1.5 mIU/L might be particularly beneficial for women with positive anti-thyroid antibodies undergoing assisted reproductive technology procedures. The analysis identifies several factors that modify the thyroid-infertility relationship including the degree of thyroid-stimulating hormone elevation, the titer of anti-thyroid antibodies, the presence of other autoimmune conditions, maternal age, and baseline ovarian reserve status, suggesting that individualized risk assessment and treatment strategies may be necessary rather than uniform protocols. Picture 2 presents treatment outcome data comparing different thyroid-stimulating hormone optimization targets in women undergoing fertility treatment.



Picture 2. Fertility Treatment Outcomes by Thyroid-Stimulating Hormone Optimization Level

The combined results point to the conclusion that thyroid autoimmunity testing ought to be incorporated into the standard fertility evaluation rules, with the testing of thyroid-stimulating hormone, free thyroxine, and anti-thyroid peroxidase antibodies being suggested for all women coming with infertility problems or going for assisted reproductive technology [5, 11]. The therapy should include both the medical side of things by providing hormonal optimization via appropriate levothyroxine dosing and also taking into account the immunological aspect, since there are studies that are pointing out the possible gains from the use of adjunctive treatments like selenium supplementation, vitamin D optimization, and in some cases, low-dose corticosteroids or IV immunoglobulin therapy, although the latter need to be supported with more evidence through randomized controlled trials [9, 12]. The study states that the best treatment is to have constant monitoring throughout the whole process of infertility treatment and during pregnancy, because the need for thyroid hormone usually increases and thus the inadequate levothyroxine dosing leads to difficulties such as miscarriage, preterm birth, and even retardation in the development of the baby's brain [3, 8]. The combination of the endocrinological and immunological view points gives a holistic view of the problem of infertility in women with thyroid autoimmunity and therefore it backs the execution of multidisciplinary management approaches that take care of both thyroid optimization and enhancement of reproduction through the involvement of reproductive endocrinologists, thyroidologists, and immunologists in care where needed.

CONCLUSION

This in-depth investigation identifies autoimmune thyroid disorders as major factors leading to female infertility via the domino effect of interlinked hormonal and immunological actions that hinder reproductive function at various natures like ovarian reserve, oocyte quality, endometrial receptivity, and capacity for pregnancy maintenance. The compilation of evidences indicates that the thyroid autoimmunity which is demonstrated either by high thyroid-stimulating

hormone levels or by the positivity of anti-thyroid antibodies, significantly affects the fertility negatively with the quantifiable declines in the ratio of conception at the range of 30-45% and the 50-100% increase in the rate of miscarriages when compared to euthyroid antibody-negative women. The complexity of this relationship is the pathophysiological nature that goes beyond just thyroid hormone deficiency to autoimmune dysregulation which produces systemic inflammation conditions adversely affecting reproductive tissues through the hypothesis of cytokine-mediated mechanisms and immune cell dysfunction. The impact on clinical practice drawn from these discoveries calls for the incorporation of thyroid function evaluation into the infertility diagnosis process as standard, with a recommendation for thyroid-stimulating hormone, free thyroxine, and anti-thyroid peroxidase antibodies assessments for each and every woman coming with a fertility-related issue. Therapeutic strategies centered on achieving thyroid-stimulating hormone optimization below 2,5 mIU/L, and potentially below 1,5 mIU/L in assisted reproductive technology contexts, demonstrate significant improvements in fertility outcomes with approximately 30-40% enhancement in conception rates and 40-50% reduction in pregnancy loss. The evidence supports early identification and aggressive treatment of thyroid dysfunction in women planning conception, with levothyroxine supplementation representing a safe, effective, and relatively inexpensive intervention that substantially improves reproductive outcomes when appropriately implemented. The immunological dimension of thyroid autoimmunity presents both challenges and opportunities, with emerging therapeutic approaches targeting immune dysregulation showing promise but requiring further validation through rigorous clinical trials before routine clinical implementation. The management of infertile women with thyroid autoimmunity benefits from multidisciplinary approaches that integrate endocrinological expertise with reproductive medicine specialization, ensuring comprehensive assessment and optimization of both thyroid function and reproductive capacity.

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