TASHKENT MEDICAL ACADEMY

Journal of Educational and Scientific Medicine

Issue 6 | 2024

OAK.uz

Google Scholar

ISSN: 2181-3175

Journal of Education & Scientific Medicine

ISSN 2181-3175

Tashkent Medical Academy

Research Article

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Features of Folliculogenesis in Women with Endocrine Infertility

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ABSTRACT

Background. *Endocrine infertility is defined as a woman's inability to conceive within one year of regular unprotected sexual intercourse, primarily caused by endocrine system disorders.*

Aim. *To study the characteristics of folliculogenesis in women with infertility caused by various endocrine pathologies and to develop more effective diagnostic and treatment methods.*

Materials and methods. *The study included 90 women with endocrine infertility, divided into three groups: Group I – hypothyroidism (n=30), Group II – hyperprolactinemia (n=30), Group III – hyperandrogenism (n=30). A control group comprised 30 healthy women. Hormonal analysis and transvaginal ultrasound were conducted to assess the number and size of antral follicles. Statistical analysis was performed using the t-test and one-way ANOVA.*

Results. *The mean number of antral follicles in women with endocrine infertility was significantly lower compared to the control group: for women with hypothyroidism – 4.5±1.2, with hyperprolactinemia – 5.1±1.3, and with hyperandrogenism – 6.8±1.4 ($p<0.05$). The average follicle diameter was also smaller: for women with hypothyroidism – 6.2±0.8 mm, with hyperprolactinemia – 6.7±0.9 mm, and with hyperandrogenism – 7.5±1.1 mm ($p<0.05$). Hormonal studies revealed significant differences in the levels of LH, FSH, prolactin, estradiol, progesterone, testosterone, T3, T4, and TSH between the study groups and the control group.*

Conclusion. *Endocrine disorders play a key role in disrupting folliculogenesis and can lead to infertility. A differentiated approach to the diagnosis and treatment of endocrine infertility, taking into account the specific hormonal profiles of each patient, may increase the effectiveness of therapy and improve reproductive outcomes for women. Future studies should include larger and more diverse samples to confirm these findings.*

Key words: *folliculogenesis, endocrine infertility, hypothyroidism, hyperprolactinemia, hyperandrogenism, hormones.*

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INTRODUCTION

Endocrine infertility, defined as a woman's inability to conceive within one year of active sexual life without the use of contraceptives, is attributed to dysfunctions within the endocrine system. The primary causes include disorders of the thyroid gland, pituitary gland, and adrenal glands, leading to hormonal imbalances and disruptions in ovulation and folliculogenesis [1].

In recent years, the prevalence of endocrine infertility has been rising, largely due to worsening environmental conditions, increased stress levels, and lifestyle changes [2].

Recent studies indicate that dietary changes, reduced physical activity, and heightened exposure to endocrine disruptors may contribute to the increasing rate of endocrine infertility [3].

Folliculogenesis is a complex process of follicle maturation within the ovaries that plays a critical role in female fertility. This process begins with the activation of primordial follicles and concludes with the formation of a dominant follicle ready for ovulation. Each stage is tightly regulated by hormones from the pituitary and ovaries. Disruptions at any stage of folliculogenesis can lead to various forms of infertility, ranging from anovulation to insufficient oocyte functionality [4].

Comparisons with international data confirm that endocrine disorders are key contributors to the pathogenesis of folliculogenesis and infertility [5].

Endocrine disorders caused by thyroid, pituitary, or adrenal dysfunctions significantly impact folliculogenesis processes. These changes may manifest as anovulation, reduced numbers of antral follicles, and decreased oocyte quality, as supported by findings from recent studies. Pituitary dysfunction leads to disrupted secretion of pituitary hormones, thereby hindering normal follicle maturation. Adrenal dysfunction can result in excessive androgen production, adversely affecting the ovarian cycle and folliculogenesis [6].

Despite numerous studies, the specific mechanisms through which various endocrine pathologies affect folliculogenesis remain inadequately understood. Subclinical hypothyroidism's impact on folliculogenesis has been less thoroughly investigated than manifest forms of the disease. Combined effects of endocrine disorders, such as hypothyroidism and hyperandrogenism, require further investigation [7]. Comparative analysis of international data reveals that such endocrine disorders represent a global challenge, as evidenced by research from Europe, Asia, and North America [8]. This highlights the need for a deeper understanding and the development of effective treatment methods that consider the unique characteristics of each population [9].

Investigating the characteristics of folliculogenesis in women with different endocrine pathologies is essential to expanding knowledge in this field and developing new, more effective methods for diagnosing and treating endocrine infertility. Ultimately, this will increase the chances for women to achieve successful conception and give birth to healthy children, which is the primary goal of reproductive medicine [10].

The aim of this study is to examine the characteristics of folliculogenesis in women with infertility due to various endocrine pathologies and to develop more effective methods for diagnosis and treatment.

MATERIALS AND METHODS

The study included 90 women with endocrine infertility. Based on the type of endocrine infertility, the patients were divided into three groups: Group I — 30 women with hypothyroidism, Group II — 30 women with hyperprolactinemia, and Group III — 30 women with hyperandrogenism. The control group consisted of 30 healthy women with no endocrine disorders or fertility issues.

Inclusion Criteria: Women aged 20 to 40 with a diagnosed case of endocrine infertility (hypothyroidism, hyperprolactinemia, or hyperandrogenism) and without other forms of infertility. Exclusion Criteria: Presence of somatic diseases affecting fertility, use of contraceptives in the past six months, and refusal to participate in the study. Hormonal assessments to determine levels of LH, FSH, prolactin, estradiol, progesterone, testosterone, T3, T4, and TSH were conducted using enzyme-linked immunosorbent assay (ELISA). The following reagent kits were used: for LH and FSH — Beckman Coulter, for prolactin — Roche Diagnostics, for estradiol and progesterone — Siemens Healthineers, for testosterone — Abbott Laboratories, for T3 and T4 — BioRad, and for TSH — BioMerieux.

Ultrasound examinations (US) included transvaginal US to measure the number and size of antral follicles and US of the thyroid gland, pituitary, and adrenal glands to assess the condition of these organs. The study was conducted on a Voluson E8 ultrasound machine (GE Healthcare) using a 7.5 MHz transvaginal probe and a 10 MHz linear probe. Folliculogenesis was assessed by the number and size of antral follicles, using data from transvaginal US performed on the Voluson E8 (GE Healthcare) with a 7.5 MHz probe. Measurements were taken at the beginning of the follicular phase (days 3-5 of the menstrual cycle). The study was conducted in the form

of a cohort analysis with longitudinal observation. For statistical data analysis, descriptive and inferential statistical methods were used. Variable distribution was checked for normality using the Shapiro-Wilk test. Comparison of mean values between groups was performed using an independent t-test and one-way analysis of variance (ANOVA). Correlation analysis was conducted using Pearson's correlation coefficient. All statistical analyses were performed using SPSS software version 25.0 (IBM Corp.).

RESULTS

The mean age of the study participants was 32.4±5.3 years. The mean body mass index (BMI) was 24±3 kg/m². Patients represented various socioeconomic backgrounds, taking into account occupational status and education level. Transvaginal US revealed that the mean number of antral follicles in women with endocrine infertility differed significantly from that in the control group. In Group I, the mean number of antral follicles was 4.5±1.2, which was significantly lower than in the control group (8.3±1.5; p<0.01). In Group II, the mean number of antral follicles was 5.1±1.3, and in Group III — 6.8±1.4, which were also significantly lower than the control values (p<0.05 and p<0.01, respectively).

The number of antral follicles is a key marker of ovarian reserve and fertility in women. The results of our study indicate a significant reduction in this marker in women with endocrine disorders compared to healthy women. In particular, women with hypothyroidism demonstrated the most pronounced reduction in the number of antral follicles. In Group I, the mean number of antral follicles was 4.5±1.2, which is 1.8 times lower than in the control group (8.3±1.5; p<0.01) (Fig. 1).

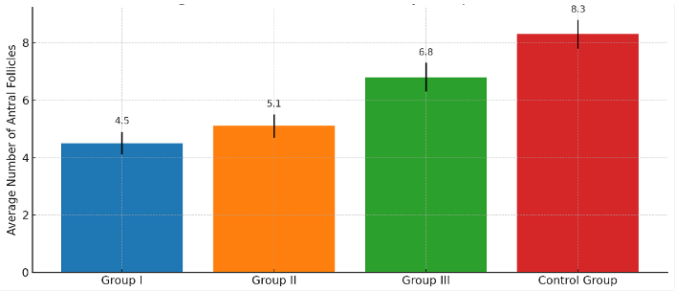

Figure 1. Average Number of Antral Follicles in Women with Different Endocrine Disorders

Similarly, women with hyperprolactinemia showed a decrease in the number of antral follicles. In Group II, the mean number of antral follicles was 5.1±1.3, 1.6 times lower than in the control group (p<0.01). Elevated prolactin levels suppress gonadotropin secretion, leading to decreased ovarian reserve and impaired follicle maturation. Patients with hyperandrogenism showed intermediate values in the number of antral follicles compared to the control group and the other study groups. In Group III, the average number of antral follicles was 6.8±1.4, which is 1.2 times lower than in the control group (p < 0.05 and p<0.01, respectively). Hyperandrogenism, often associated with adrenal dysfunction, leads to disruptions in follicle development and maturation, as confirmed by our data.

The size of antral follicles also varied across the studied groups. In women with hypothyroidism, the average follicle diameter was 6.2±0.8 mm, which is significantly smaller than in the control group (9.1±1.0 mm; p<0.01). In patients with hyperprolactinemia, this parameter was 6.7±0.9 mm, and in women with hyperandrogenism — 7.5±1.1 mm, both of which were smaller than in the control group (p<0.05 and p<0.01, respectively) (Fig. 2).

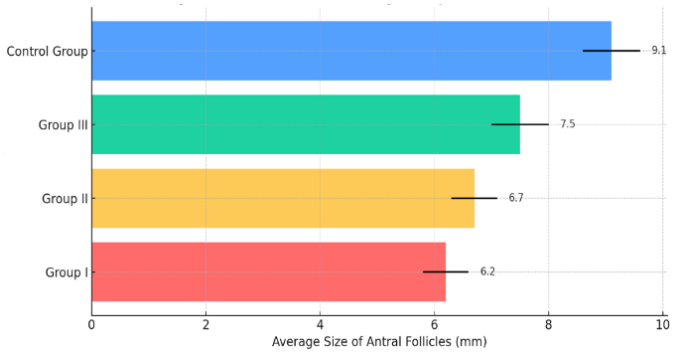

Figure 2. Average Size of Antral Follicles in Women with Various Endocrine Disorders

The size of antral follicles is a critical indicator of their maturity and readiness for ovulation. In our study, the most pronounced deficit in antral follicle size was observed in women with hypothyroidism. The mean follicle diameter in Group I was 6.2±0.8 mm, which is 1.5 times smaller than that of the control group (9.1±1.0 mm; p<0.01). Women with hyperprolactinemia also showed a reduction in antral follicle size. The mean follicle diameter in Group II was 6.7±0.9 mm, 1.4 times smaller than the control group (p<0.05 and p<0.01, respectively). Elevated prolactin levels disrupt normal gonadotropin function, leading to insufficient follicular development. Women with hyperandrogenism exhibited smaller follicle sizes than the control group, though these changes were less pronounced than in the other groups. The mean follicle size in Group III was 7.5±1.1 mm, 1.2 times smaller than in the control group (p<0.05 and p < 0.01, respectively). Hyperandrogenism, often associated

with excess androgen production, may disrupt the normal ovarian cycle, leading to suboptimal follicle development. Our findings confirm that endocrine disorders significantly impact the number and size of antral follicles in women suffering from infertility. These results highlight the necessity for a personalized approach in diagnosing and treating endocrine infertility, which could enhance reproductive outcomes in affected patients.

Hormonal analyses revealed substantial differences in levels of LH, FSH, prolactin, estradiol, progesterone, testosterone, T3, T4, and TSH among the study groups and the control group. In Group I, LH levels were 4.2±1.1 mIU/mL, significantly lower than in the control group (6.1±1.2 mIU/mL; p<0.01). FSH was also reduced, at 5.3±1.4 mIU/mL versus 7.2±1.5 mIU/mL in the control group (p<0.01). TSH levels were significantly elevated, measuring 5.6±2.0 μIU/mL, more than twice the control levels (2.5±0.8 μIU/mL; p<0.01). T3 and T4 levels were markedly reduced, at 1.1±0.2 ng/mL and 6.4±1.5 μg/dL, respectively, compared to the control group (1.8±0.3 ng/mL and 8.2±1.7 μg/dL; p<0.01). These data confirm the presence of hypothyroidism, which adversely affects the hypothalamic-pituitary-ovarian axis, leading to decreased levels of gonadotropins and thyroid hormones essential for normal folliculogenesis and ovulation.

In Group II, the prolactin level was 45.6±8.3 ng/mL, more than three times higher than the control value (13.4±2.1 ng/mL; p<0.01). LH and FSH levels were also altered, measuring 3.8±0.9 mIU/mL and 4.9±1.2 mIU/mL, respectively, which were significantly lower compared to the control group (6.1±1.2 mIU/mL and 7.2±1.5 mIU/mL; p<0.05). Elevated prolactin levels inhibit gonadotropin secretion, leading to hypogonadism and anovulation. These findings align with previous research, indicating that hyperprolactinemia is a leading cause of endocrine infertility. In Group III, the testosterone level was 2.8±0.7 ng/mL, over four times the control value (0.7±0.2 ng/mL; p<0.01). LH and FSH levels were also elevated, measuring 6.3±1.5 mIU/mL and 7.2 ± 1.7 mIU/mL, respectively, which were higher compared to the control group (6.1±1.2 mIU/mL and 7.2±1.5 mIU/mL; p<0.05) (see Table 1).

This table clearly illustrates the differences in hormone levels among the groups and the control group. Hyperandrogenism, often associated with adrenal dysfunction, leads to increased androgen levels, which negatively impact normal ovarian function and folliculogenesis. Elevated LH and FSH levels in this group may represent a compensatory response to the high androgen levels. Correlation analysis showed significant negative correlations between TSH levels and the number of antral follicles in Group I (r=-0.62; p<0.01), as well as between prolactin levels and the number of antral follicles in Group II (r=-0.54; p<0.01). In Group III, a significant negative correlation was found between testosterone levels and antral follicle size (r=-0.48; p<0.05) (Figure 3).

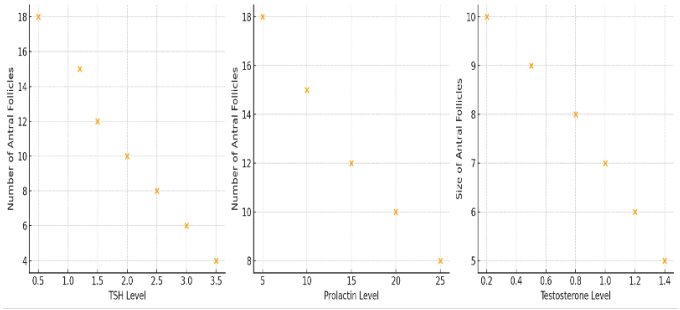

Figure 3. Correlation between hormone levels and the number/size of antral follicles across different groups

Comparison of results across study groups indicated that women with hypothyroidism and hyperprolactinemia exhibited more pronounced changes in both the number and size of antral follicles compared to women with hyperandrogenism (p<0.05). This underscores the need for a differentiated approach in diagnosing and treating endocrine infertility, taking into account the specific hormonal profiles of each patient. Elevated TSH levels (5.6±2.0 μIU/mL) and reduced T3 (1.1±0.2 ng/mL) and T4 (6.4±1.5 μg/dL) levels indicate insufficient stimulation of the hypothalamic-pituitary-ovarian axis. This leads to reduced LH (4.2±1.1 mIU/mL) and FSH (5.3±1.4 mIU/mL) levels, resulting in a decrease in both the number (4.5±1.2) and size (6.2 ± 0.8 mm) of antral follicles (p<0.01). These findings align with previous studies that highlight the crucial role of thyroid hormones in regulating reproductive function.

Prolactin levels in Group II (45.6±8.3 ng/mL) were over three times higher than in the control group (13.4±2.1 ng/mL; p<0.01). High prolactin levels inhibit LH (3.8±0.9 mIU/mL) and FSH (4.9±1.2 mIU/mL) secretion, leading to a reduction in the number (5.1±1.3) and size (6.7±0.9 mm) of antral follicles (p<0.05). These findings are consistent with previous research showing that hyperprolactinemia is a leading cause of ovulatory disorders and infertility. Women with hyperandrogenism showed intermediate values in both the number and size of antral follicles compared to the control group and other study groups. Testosterone levels in Group III (2.8±0.7 ng/mL) were more than four times higher than

Table 1. Hormonal Indicators in Examined Women with Endocrine Disorders, M±m

Group	LH (mIU/mL)	FSH (mIU/mL)	TTH (µIU/mL)	T3 (ng/mL)	T4 (µg/dL)	Prolactin (ng/mL)	Testosterone (ng/mL)
Group I, n=30	4.2±1.1*	5.3± 1.4**	5.6 ± 2.0	1.1 ± 0.2	6.4 ± 1.5	-	-
Group II, n=30	3.8±0.9*	4.9± 1.2**	-	-	-	45.6 ± 8.3	-
Group III, n=30	6.3±1.5*	7.2 ± 1.7*	-	-	-	-	2.8 ± 0.7
Control Group	6.1 ± 1.2	7.2 ± 1.5	2.5 ± 0.8	1.8 ± 0.3	8.2 ± 1.7	13.4 ± 2.1	0.7 ± 0.2

Note: * $p<0.05$, ** $p<0.01$- Significant difference compared to the control group

the control value (0.7±0.2 ng/mL; $p<0.01$). Elevated androgen levels negatively impact the ovarian cycle, reducing both the number (6.8±1.4) and size (7.5±1.1 mm) of antral follicles ($p<0.05$). This confirms the adverse effect of hyperandrogenism on folliculogenesis and reproductive function.

A multivariate analysis was conducted to identify more subtle relationships between variables. The results showed that TSH and prolactin levels significantly influence the number of antral follicles ($p<0.01$), while testosterone levels substantially affect follicle size ($p<0.05$). These data emphasize the importance of hormonal imbalances in disrupting folliculogenesis in women with endocrine infertility. These results underscore the need for an individualized approach in diagnosing and treating endocrine infertility, which considers each patient's unique hormonal profile. Early detection and correction of hormonal disorders can significantly enhance fertility and increase the chances of successful conception.

DISCUSSION

Our findings confirm that endocrine disorders significantly impact folliculogenesis in women with infertility. Data analysis revealed that various endocrine pathologies lead to notable changes in the number and size of antral follicles as well as in hormonal profiles [3, 5, 7].

In Group I, patients with hypothyroidism showed a significant reduction in the quantity and size of antral follicles compared to the control group, corroborating previous studies that indicate hypothyroidism negatively affects the ovulatory cycle by reducing LH and FSH levels, thus impeding follicle maturation. Hormone tests showed elevated TSH and reduced T3 and T4 levels, reinforcing hypothyroidism as a primary factor affecting folliculogenesis). In Group II, women with hyperprolactinemia also displayed reduced numbers and sizes of antral follicles. Elevated prolactin levels inhibit LH and FSH secretion, leading to anovulation and disrupted folliculogenesis. Our findings align with previous research identifying hyperprolactinemia as a leading cause of endocrine infertility. Group III, comprising women with hyperandrogenism, exhibited increased testosterone levels, which corresponded to a reduction in the quantity and size of antral follicles. Hyperandrogenism, frequently associated with polycystic ovary syndrome, disrupts the normal ovulatory cycle, often resulting in hyperandrogenic anovulation. Our results confirm that high androgen levels adversely affect folliculogenesis [3].

Correlation analysis demonstrated significant negative correlations between TSH levels and the number of antral follicles in Group I, prolactin levels and antral follicle count in Group II, and testosterone levels and follicle size in Group III. These findings highlight a direct link between hormonal imbalances and disruptions in folliculogenesis, emphasizing the need for targeted hormonal treatments for each patient group. Intergroup comparisons revealed that women with hypothyroidism and hyperprolactinemia experienced more pronounced disruptions in folliculogenesis than those with hyperandrogenism, underscoring the importance of a differentiated approach to diagnosing and treating endocrine infertility based on specific hormonal irregularities. The clinical implications of our findings underscore the necessity of early diagnosis and appropriate therapy for endocrine disorders to enhance folliculogenesis and improve chances of successful conception. Individualized treatment protocols aimed at correcting specific hormonal imbalances can markedly improve reproductive outcomes in women with endocrine infertility [2].

For example, TSH correction with levothyroxine in patients with hypothyroidism can enhance ovulatory cycles and increase antral follicle counts. Cabergoline use in hyperprolactinemic women can normalize LH and FSH levels, promoting follicle maturation. For hyperandrogenic patients, reducing androgen levels with spironolactone may improve both the quantity and quality of antral follicles [1].

Our study has several limitations. First, the sample size and geographic scope are limited, potentially affecting the generalizability of the results. Future studies should include larger and more diverse samples to validate our conclusions. Second, the study's timeframe (one year) may not fully capture the long-term effects of endocrine disorders on folliculogenesis. Extended studies are needed to assess the durability of these results. Lastly, using only one method to assess folliculogenesis (transvaginal ultrasound) may limit measurement accuracy, so future research should consider additional imaging methods and oocyte quality assessments [7].

Further research is required to elucidate the molecular mechanisms by which various endocrine pathologies affect folliculogenesis. Additionally, new diagnostic and therapeutic approaches should be developed to address specific hormonal imbalances in women with diverse endocrine disorders. Special attention should be paid to studying the combined effects of multiple endocrine disturbances on reproductive function. Our study confirms that endocrine disorders significantly influence folliculogenesis in infertile women. Depending on the type of endocrine disorder, different alterations are observed in the number and size of antral follicles, as well as in hormonal profiles. These findings underscore the need for a differentiated approach to diagnosing and treating endocrine infertility, which could increase treatment efficacy and improve reproductive outcomes [5].

CONCLUSION

Our study demonstrated that endocrine disorders, such as hypothyroidism, hyperprolactinemia, and hyperandrogenism, profoundly impact folliculogenesis in infertile women. Each disorder—hypothyroidism, pituitary dysfunction, and adrenal dysfunction—uniquely affects follicle maturation. In hypothyroid women, antral follicle counts were reduced by 1.8 times compared to the control group; in hyperprolactinemic women by 1.6 times; and in hyperandrogenic women by 1.2 times. Antral follicle size also varied: in hypothyroid patients, follicle diameter was 1.5 times smaller; in hyperprolactinemic patients, 1.4 times smaller; and in hyperandrogenic patients, 1.2 times smaller. Hormone tests revealed that hypothyroid women had reduced LH and FSH levels with TSH elevated by over twofold, and reduced T3 and T4 levels. Hyperprolactinemic women had prolactin levels three times higher than normal, with reduced LH and FSH levels, while hyperandrogenic women had testosterone levels over fourfold higher than the control.

Correlation analysis indicated that TSH and prolactin negatively correlate with antral follicle count, and testosterone negatively correlates with follicle size. These findings highlight the importance of an individualized approach to diagnosing and treating endocrine infertility. Hormonal correction can significantly enhance reproductive outcomes. Future studies should account for these factors and include larger, more varied samples. Thus, endocrine disorders play a critical role in folliculogenesis disruptions, often leading to infertility. A differentiated approach to diagnosing and treating endocrine infertility can increase treatment efficacy and improve reproductive outcomes in women.

Ethics approval and consent to participate - All patients gave written informed consent to participate in the study.

Consent for publication - The study is valid, and recognition by the organization is not required. The author agrees to open publication

Availability of data and material - Available

Competing interests - No

Financing – No financial support has been provided for this work

Conflict of interests - The authors declare that there is no conflict of interest.

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How to Cite: *Anvarova Sh.A., Shukurov F.I., Ismoilova Sh.I. Features of folliculogenesis in women with endocrine infertility // Journal of Educational & Scientific Medicine, 2024. Vol. 1, Issue 6, P. 106–113.*

ENDOKRIN GENEZLI BEPUSHT AYOLLARDA FOLLIKULOGENEZNING JARAYONINING XUSUSIYATLARI

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Toshkent tibbiyot akademiyasi

ABSTRAKT

Dolzarbligi. Endokrin bepushtlik — ayolning bir yil davomida kontratseptivlarsiz faol jinsiy hayot olib borgan holda homilador bo‘lishga qodir emasligi bilan tavsiflanadi va endokrin tizimdagi buzilishlar bilan bog‘liqdir.

Maqsad. Turli endokrin patologiyalar bilan bog‘liq bepusht ayollarda follikulogenez xususiyatlarini o‘rganish va tashxislash hamda davolashning samaraliroq usullarini ishlab chiqish.

Material va usullar. Tadqiqotga endokrin bepushtlik aniqlangan 90 nafar ayol jalb qilindi, ular uchta guruhga ajratildi: I guruh — gipotireoz kasalligi bilan (n=30), II guruh — giperprolaktinemiya kasalligi bilan (n=30), III guruh — giperandrogeniya kasalligi bilan (n=30). Nazorat guruhi 30 sog‘lom ayoldan iborat. Gormonal tekshiruv va antral follikullar soni va hajmini baholash uchun transvaginal ultratovush tekshiruvi o‘tkazildi. Statistik tahlil t-test va bir faktorli dispersion tahlil (ANOVA) yordamida amalga oshirildi.

Natijalar. Endokrin bepushtlik aniqlangan ayollarda antral follikullar soni nazorat guruhiga nisbatan ancha kam ekanligi aniqlandi: gipotireoz bilan kasallangan ayollarda — 4,5±1,2, giperprolaktinemiyaga ega ayollarda — 5,1±1,3, giperandrogeniya mavjud ayollarda — 6,8±1,4 ($p<0,05$). Follikulalar diametrining o‘rtacha ko‘rsatkichi ham kichik bo‘ldi: gipotireozga ega ayollarda — 6,2±0,8 mm, giperprolaktinemiyaga mavjud ayollarda — 6,7±0,9 mm, giperandrogeniya mavjud ayollarda — 7,5±1,1 mm ($p<0,05$). Gormonal tekshiruvlar natijalari LG, FSG, prolaktin, estradiol, progesteron, testosteron, T3, T4 va TTG darajalarida tadqiqot guruhlari va nazorat guruhi o‘rtasida sezilarli farqlar mavjudligini ko‘rsatdi.

Xulosa. Endokrin buzilishlar follikulogenez jarayonining buzilishida asosiy rol o‘ynaydi va bepushtlikka olib kelishi mumkin. Endokrin bepushtlikni tashxislash va davolashda har bir bemorning o‘ziga xos gormonal profilini hisobga oladigan differensial yondashuv davolash samaradorligini oshirishi va ayollarda reproduktiv natijalarni yaxshilashi mumkin.

Kalit so‘zlar: follikulogenez, endokrin bepushtlik, gipotireoz, giperprolaktinemiya, giperandrogeniya, gormonlar.

ОСОБЕННОСТИ ФОЛЛИКУЛОГЕНЕЗА У ЖЕНЩИН С БЕСПЛОДИЕМ ЭНДОКРИННОГО ГЕНЕЗА

Анварова Ш.А., Шукуров Ф.И., Исмоилова Ш.И.
Ташкентская медицинская академия

АБСТРАКТ

Актуальность. Эндокринное бесплодие определяется как неспособность женщины зачать ребенка в течение одного года активной половой жизни без использования контрацептивов и обусловлено нарушениями в эндокринной системе.

Цель. Изучение особенностей фолликулогенеза у женщин с бесплодием, обусловленным различными эндокринными патологиями, и разработка более эффективных методов диагностики и лечения.

Материалы и методы. Исследование включало 90 женщин с эндокринным бесплодием, разделенных на три группы: I группа — гипотиреоз (n=30), II группа — гиперпролактинемия (n=30), III группа — гиперандрогения (n=30). Контрольная группа состояла из 30 здоровых женщин. Проводилось гормональное исследование и трансвагинальное ультразвуковое исследование для оценки количества и размера антральных фолликулов. Статистический анализ проводился с использованием t-теста и однофакторного дисперсионного анализа (ANOVA).

Результаты. Среднее количество антральных фолликулов у женщин с эндокринным бесплодием было значительно ниже по сравнению с контрольной группой: у женщин с гипотиреозом — 4,5±1,2, у женщин с гиперпролактинемией — 5,1±1,3, у женщин с гиперандрогенией — 6,8±1,4 ($p < 0,05$). Средний диаметр фолликулов также был меньше: у женщин с гипотиреозом — 6,2±0,8 мм, у женщин с гиперпролактинемией — 6,7±0,9 мм, у женщин с гиперандрогенией — 7,5±1,1 мм ($p < 0,05$).

Заключение. Эндокринные нарушения играют ключевую роль в нарушении фолликулогенеза и могут приводить к бесплодию. Дифференцированный подход к диагностике и лечению эндокринного бесплодия, учитывающий специфические гормональные профили каждой пациентки, может повысить эффективность терапии и улучшить репродуктивные исходы у женщин.

Ключевые слова: фолликулогенез, эндокринное бесплодие, гипотиреоз, гиперпролактинемия, гиперандрогения, гормоны