Volume 3, Issue 12, December 2025 ISSN (E): 2938-3765

PATHOGENETIC ASPECTS OF OVARIAN POLYCYSTOSIS SYNDROME IN PATIENTS WITH METABOLIC DISORDERS

Urunova Farangiz
Resident of the Master's Program 1
Course of the Department of Endocrinology

Abdullaeva Raykhon
1st Year Clinical Resident

Rasulova Mokhinur
1st Year Clinical Resident; Scientific Supervisor: PhD,
Associate Professor, Head of the Department of Endocrinology

Negmatova G. Sh.
Samarkand State Medical University
Samarkand, Uzbekistan

Abstract
Polycystic ovary syndrome (PCOS) is a common hormonal disorder that occurs most often in women of reproductive age. The main signs of ovarian polycystic disease include menstrual cycle disorders, irregular ovulation, high levels of androgen hormones in the blood, and a specific polycystic structure of the ovaries according to ultrasound results [1,6].

Keywords: Polycystic ovary, metabolic disorders, pathogenesis, diagnosis, follicles, obesity.

Introduction

Polycystic ovary syndrome is the most common endocrine disease in women and the main cause of anovulatory infertility. Since the symptoms of the disease are extremely diverse, the diagnosis of the syndrome can be difficult. To date, Rotterdam diagnostic criteria for this condition have been adopted (European Society of Human Reproduction and Embryology/American Society for Reproductive Medicine, 2003):

1) clinical or biochemical signs of hyperandrogenism;
2) anovulation;
3) polycystic ovaries according to ultrasound.

To make a diagnosis, two out of three criteria are sufficient [11,12,13].

Considering the widespread prevalence of ovarian polycystic syndrome among women of reproductive age (4-12%), timely detection of metabolic syndrome manifestations in them could contribute to improving both the general and reproductive health of women [14].

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

More and more evidence is emerging that TPS affects the entire life: starting in the mother's womb in genetically predisposed women, it manifests clinically during puberty and continues throughout the reproductive period.

Patients with PCOS are at higher risk of developing cardiovascular diseases, hypertension, diabetes mellitus, and other metabolic complications, especially after menopause [15].

In reproductive age, the disease can lead to anovulatory infertility and provoke the development of gestational complications (abortion, gestational diabetes, and preeclampsia) [1, 4,8,9]. Therefore, early diagnosis of PCOS is crucial, and only then will the patient be carefully monitored to reduce the risk of complications.

The appearance of polycystic ovary syndrome is associated with many factors, including both genetic predisposition and external influences. The main links of the pathology are disorders in the regulation of the hormonal background, in particular, the imbalance between the level of insulin and sex hormones [1,2,3].

An excessive level of insulin has a stimulating effect on the secretion of androgens in the ovaries, which leads to delayed maturation of follicles and their polycystic changes. In addition, environmental factors, stress, and the presence of obesity play an important role [4,5,6].

The pathogenesis of polycystic ovary syndrome is linked to a complex of endocrine and metabolic pathologies. Disruptions in the regulation of the hypothalamic-pituitary-ovarian axis lead to increased secretion of luteinizing hormone (LH) compared to follicle-stimulating hormone (FSH), which provokes excessive production of androgens [7,8,9].

High insulin levels due to insulin resistance contribute to increased hyperandrogenic processes and impaired normal follicular growth. As a result, a condition that was first described as Stein-Leventhal syndrome more than half a century ago develops [10,11].

Understanding the pathogenesis serves as a foundation for developing effective treatment regimens and allows for targeted intervention in hormonal and metabolic imbalances. The mechanisms of PCOS development are being actively studied at the level of the hypothalamic-pituitary complex, ovaries, adrenal glands, and adipose tissue. It has been shown that various environmental factors, namely low socio-economic standards and an unhealthy lifestyle (smoking, overeating, lack of physical activity), can contribute to the development of polycystic ovary syndrome [12,13].

However, genetic predisposition is given special importance in the etiopathogenesis of PCOS. The risk of developing the disease increases by 30-50% in patients with a family history of PCOS [3,5,6]. The patterns of inheritance of this disease have not been definitively studied, however, considering the clinical and laboratory heterogeneity, the syndrome most likely has a polygenic or multifactorial type of inheritance.

Candidate genes can be combined into pathogenetic groups: 1) genes involved in the synthesis and action of steroid hormones; 2) genes responsible for the synthesis and regulation of pituitary hormones; 3) genes responsible for the synthesis and action of insulin; 4) genes regulating body weight; 5) genes encoding inflammation mediators; 6) genes of the main complex of histocompatibility [1,3,4,5].

The molecular mechanism of PCOS is a complex interaction of genetic predispositions, hyperinsulinemia/insulin resistance, hormonal imbalances (hyperandrogenism), and chronic inflammation that leads to impaired folliculogenesis, anovulation, cyst formation in the ovaries, and

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

metabolic problems affecting insulin signaling pathways and steroidogenesis. The main role is played by changes in the expression of estrogen receptors (ERα/ERβ), resistance to progesterone, and an increase in the level of androgens, which disrupts the normal cycle of egg development.

Predisposition to PCOS development is possible not only in the female line but also in the male line, when male relatives may experience early baldness, decreased concentration of sex steroid-binding globulin (SSBG), and insulin resistance (IR) [13].

PCOS often progresses to hyperandrogenism (HA). At the same time, HA occurring in PCOS accounts for 50-80% of all forms of hyperandrogenic conditions. Hyperandrogenic conditions are characterized by pathological disorders caused by excessive exposure to androgens [3, 14].

In polycystic ovary syndrome, functional ovarian hypertension is observed. Based on modern diagnostic criteria for PCOS, various phenotypes are distinguished, namely: hyperandrogenism and chronic anovulation; hyperandrogenism and polycystic ovaries according to ultrasound data with preserved ovulatory cycles; chronic anovulation and polycystic ovaries without hyperandrogenism; hyperandrogenism, chronic anovulation, and polycystic ovaries [1, 4,9].

Thus, the conducted analysis of literature data indicates that persistent hypo- or anovulation in women with PCOS, who often have hyperinsulinemia and hyperandrogenism, leads to relatively constant circulating levels of estradiol comparable to the early follicular phase.

Moderately elevated estradiol levels occur due to increased peripheral conversion of androstendion to estrone in adipose tissue.

Additionally, insulin-like growth factors (IGF) and their binding proteins are regulated and affect endometrial cell components, while hyperinsulinemia regulates IGFBP-1, leading to an increase in free IGF-I in endometrial cells. The inflammatory environment observed in women with PCOS, through the secretion of cytokines, negatively affects the effect of insulin, exacerbating disorders of endometrial energy homeostasis.

References

1. Azziz R. PCOS: a diagnostic challenge //Reproductive biomedicine online. – 2004. – Т. 8. – №. 6. – С. 644-648.

2. Hoeger K. M., Dokras A., Piltonen T. Update on PCOS: consequences, challenges, and guiding treatment //The Journal of Clinical Endocrinology & Metabolism. – 2021. – Т. 106. – №. 3. – С. e1071-e1083.

3.Rosenfield R. L., Ehrmann D. A. The pathogenesis of polycystic ovary syndrome (PCOS): the hypothesis of PCOS as functional ovarian hyperandrogenism revisited //Endocrine reviews. – 2016. – Т. 37. – №. 5. – С. 467-520.

4.Wild R. A. Long-term health consequences of PCOS //Human reproduction update. – 2002. – Т. 8. – №. 3. – С. 231-241.

5.Kahsar-Miller M. D. et al. Prevalence of polycystic ovary syndrome (PCOS) in first-degree relatives of patients with PCOS //Fertility and sterility. – 2001. – Т. 75. – №. 1. – С. 53-58.

6.Pasquali R. et al. PCOS Forum: research in polycystic ovary syndrome today and tomorrow //Clinical endocrinology. – 2011. – Т. 74. – №. 4. – С. 424-433.

7.Duleba A. J., Dokras A. Is PCOS an inflammatory process? //Fertility and sterility. – 2012. – Т. 97. – №. 1. – С. 7-12.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

8. Diamanti-Kandarakis E., Kandarakis H., Legro R. S. The role of genes and environment in the etiology of PCOS //Endocrine. – 2006. – Т. 30. – №. 1. – С. 19-26.
9. Khan M. J., Ullah A., Basit S. Genetic basis of polycystic ovary syndrome (PCOS): current perspectives //The application of clinical genetics. – 2019. – С. 249-260.
10. Yang J., Chen C. Hormonal changes in PCOS //Journal of Endocrinology. – 2024. – Т. 261. – №. 1.
11. De Leo V. et al. Genetic, hormonal and metabolic aspects of PCOS: an update //Reproductive Biology and Endocrinology. – 2016. – Т. 14. – №. 1. – С. 1-17.
12. Ajmal N., Khan S. Z., Shaikh R. Polycystic ovary syndrome (PCOS) and genetic predisposition: A review article //European journal of obstetrics & gynecology and reproductive biology: X. – 2019. – Т. 3. – С. 100060.
13. Homburg R. Pregnancy complications in PCOS //Best Practice & Research Clinical Endocrinology & Metabolism. – 2006. – Т. 20. – №. 2. – С. 281-292.
14. Patel S. Polycystic ovary syndrome (PCOS), an inflammatory, systemic, lifestyle endocrinopathy //The Journal of steroid biochemistry and molecular biology. – 2018. – Т. 182. – С. 27-36.
15. Patel S. Polycystic ovary syndrome (PCOS), an inflammatory, systemic, lifestyle endocrinopathy //The Journal of steroid biochemistry and molecular biology. – 2018. – Т. 182. – С. 27-36.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5