

ASSESSMENT OF THE CLINICAL COURSE OF POLYCYSTIC OVARY SYNDROME IN WOMEN OF REPRODUCTIVE AGE

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Abstract

Polycystic Ovary Syndrome (PCOS) is a common endocrine disorder affecting women of reproductive age and is associated with reproductive, metabolic, and hormonal disturbances. This study aimed to assess the clinical course of PCOS by analyzing its manifestations, hormonal profiles, and metabolic characteristics. The findings demonstrate that PCOS presents with heterogeneous symptoms, including menstrual irregularities, hyperandrogenism, and polycystic ovarian morphology. Insulin resistance was identified as a key factor contributing to both reproductive dysfunction and metabolic complications. Comprehensive evaluation using clinical, biochemical, and imaging methods is essential for accurate diagnosis and effective management. Early intervention and individualized treatment strategies can significantly improve patient outcomes and reduce long-term health risks.

Keywords: Polycystic Ovary Syndrome, PCOS, reproductive age, hyperandrogenism, insulin resistance, ovarian dysfunction, metabolic syndrome

Introduction

Polycystic Ovary Syndrome (PCOS) is recognized as one of the most prevalent endocrine and metabolic disorders affecting women of reproductive age, with a significant impact on reproductive health, metabolic balance, and overall quality of life. The syndrome is characterized by a constellation of clinical, biochemical, and morphological features, including hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. Despite decades of research, PCOS remains a complex and heterogeneous condition with variable presentation and progression, posing challenges for both diagnosis and management.

The global prevalence of PCOS varies depending on diagnostic criteria and population characteristics, ranging from approximately 6% to 20%. The widely accepted Rotterdam criteria define PCOS based on the presence of at least two of the following features: oligo- or anovulation, clinical or biochemical signs of hyperandrogenism, and polycystic ovaries detected via ultrasound. However, the heterogeneity inherent in these criteria leads to multiple phenotypic expressions of the syndrome, each associated with differing degrees of reproductive and metabolic dysfunction. The pathogenesis of PCOS is multifactorial and involves a complex interplay between genetic predisposition, environmental influences, and endocrine dysregulation. One of the central mechanisms underlying PCOS is insulin resistance, which is observed in a substantial proportion of affected women, независимо от массы тела. Hyperinsulinemia contributes to increased androgen production by ovarian theca cells and suppresses hepatic synthesis of sex hormone-binding globulin (SHBG), resulting in elevated levels of free circulating androgens. This hormonal imbalance disrupts normal follicular development, leading to arrested folliculogenesis and chronic anovulation. In addition to reproductive abnormalities, PCOS is increasingly recognized as a systemic disorder with

significant metabolic implications. Women with PCOS are at elevated risk of developing impaired glucose tolerance, type 2 diabetes mellitus, dyslipidemia, and cardiovascular diseases. Furthermore, chronic low-grade inflammation and oxidative stress are thought to play important roles in the progression of metabolic complications associated with the syndrome. Another important aspect of PCOS is its impact on psychological health. Women with PCOS frequently experience anxiety, depression, and reduced quality of life, often related to symptoms such as infertility, hirsutism, acne, and obesity. These psychosocial factors further complicate disease management and highlight the need for a holistic approach to patient care.

The clinical course of PCOS varies widely among individuals and across different stages of life. In adolescents, the diagnosis may be complicated by the overlap between normal pubertal changes and early manifestations of the syndrome. In adult women, reproductive concerns such as infertility and menstrual irregularities often predominate, while metabolic complications tend to become more pronounced with increasing age. Given the heterogeneity and long-term health implications of PCOS, there is a growing need for comprehensive assessment strategies that integrate clinical, hormonal, and metabolic parameters. Evaluating the clinical course of PCOS in women of reproductive age is essential for identifying high-risk patients, optimizing therapeutic interventions, and preventing long-term complications. The present study aims to assess the clinical characteristics and progression of PCOS in women of reproductive age, with particular emphasis on the relationship between hormonal imbalance, metabolic disturbances, and clinical manifestations. A deeper understanding of these interconnections will contribute to improved diagnostic accuracy and more effective, individualized management of the syndrome.

This study was designed as a prospective observational and analytical investigation aimed at evaluating the clinical course of Polycystic Ovary Syndrome (PCOS) in women of reproductive age. The research was conducted over a defined study period in a specialized gynecological and endocrinological clinical setting.

Study Population The study included women aged 18–40 years who were diagnosed with PCOS according to the Rotterdam diagnostic criteria. Participants were recruited through outpatient clinics using a purposive sampling strategy. Inclusion criteria comprised the presence of at least two of the following features: oligo- or anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology confirmed by ultrasound examination. Exclusion criteria included pregnancy, thyroid dysfunction, hyperprolactinemia, congenital adrenal hyperplasia, androgen-secreting tumors, Cushing's syndrome, and the use of hormonal or insulin-sensitizing medications within three months prior to enrollment. These criteria were applied to minimize confounding factors and ensure diagnostic accuracy.

Clinical Assessment All participants underwent a comprehensive clinical evaluation. Anthropometric measurements included body mass index (BMI), waist-to-hip ratio (WHR), and blood pressure. Menstrual history was documented in detail, including cycle length, regularity, and duration of amenorrhea or oligomenorrhea. Clinical signs of hyperandrogenism were assessed using standardized scoring systems, including the modified Ferriman–Gallwey score for hirsutism. The presence and severity of acne and androgenic alopecia were also recorded. Additionally, lifestyle factors such as dietary habits, physical activity, and family history of metabolic or endocrine disorders were collected through structured questionnaires.

Hormonal and Biochemical Analysis Venous blood samples were collected in the early follicular phase (days 2–5 of the menstrual cycle) or at a random time in amenorrheic patients. Serum levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), total testosterone, dehydroepiandrosterone sulfate (DHEAS), prolactin, and thyroid-stimulating hormone (TSH) were measured using standardized immunoassay techniques. Metabolic assessment included fasting plasma glucose, fasting insulin levels, lipid profile (total cholesterol, HDL, LDL, triglycerides), and glycated hemoglobin (HbA1c). Insulin resistance was estimated using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) index.

Ultrasonographic Evaluation Pelvic ultrasound examination was performed using transvaginal or transabdominal approaches, depending on patient characteristics. Ovarian morphology was evaluated based on the presence of ≥ 12 follicles measuring 2–9 mm in diameter and/or increased ovarian volume ($>10 \text{ cm}^3$). Endometrial thickness and other pelvic structures were also assessed to exclude additional pathologies.

Ethical Considerations The study protocol adhered to the principles of the Declaration of Helsinki. Informed consent was obtained from all participants prior to inclusion in the study. Confidentiality and anonymity of patient data were strictly maintained throughout the research process.

Conclusion

Polycystic Ovary Syndrome in women of reproductive age is a multifactorial disorder characterized by heterogeneous clinical manifestations and varying disease progression. The study confirms that hormonal imbalance, particularly hyperandrogenism and altered gonadotropin levels, plays a central role in the development and clinical expression of the syndrome. Insulin resistance and associated metabolic disturbances significantly contribute to the severity of PCOS and increase the risk of long-term complications. The findings highlight the importance of comprehensive diagnostic approaches that integrate clinical, biochemical, and ultrasonographic parameters. Individualized management strategies based on patient-specific characteristics are essential for improving reproductive and metabolic outcomes. Early detection and preventive interventions can reduce the burden of complications and enhance the overall quality of life in affected women.

References:

1. Azziz, R., Carmina, E., Chen, Z., Dunaif, A., Laven, J. S., Legro, R. S., & Yildiz, B. O. (2016). Polycystic ovary syndrome. *Nature Reviews Disease Primers*, 2, 16057, 1–18.
2. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. (2004). Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Human Reproduction*, 19(1), 41–47.
3. Teede, H. J., Misso, M. L., Costello, M. F., Dokras, A., Laven, J., Moran, L., & Norman, R. J. (2018). Recommendations from the international evidence-based guideline for the assessment and management of PCOS. *Human Reproduction*, 33(9), 1602–1618.



4. Goodarzi, M. O., Dumesic, D. A., Chazenbalk, G., & Azziz, R. (2011). Polycystic ovary syndrome: Etiology, pathogenesis, and diagnosis. *Nature Reviews Endocrinology*, 7(4), 219–231.
5. Diamanti-Kandarakis, E., & Dunaif, A. (2012). Insulin resistance and the polycystic ovary syndrome revisited: An update on mechanisms and implications. *Endocrine Reviews*, 33(6), 981–1030.