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Review Article

Polycystic Ovary Syndrome (PCOS) overview: epidemiology, pathophysiology, symptoms, diagnosis, treatment, complications, and Future Directions Research

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Abstract

Polycystic ovary syndrome (PCOS) is the most prevalent endocrine and metabolic disorder affecting women of reproductive age and is characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. Its heterogeneous clinical presentation encompasses reproductive, metabolic, and psychological manifestations, making diagnosis and management challenging. The global prevalence of PCOS ranges from approximately 5% to 21%, depending on the diagnostic criteria used, with increasing incidence worldwide highlighting its significance as a major public health concern. The pathophysiology of PCOS is multifactorial, involving complex interactions among genetic susceptibility, insulin resistance, hyperinsulinemia, functional ovarian hyperandrogenism, hypothalamic–pituitary–ovarian axis dysfunction, and environmental influences. Clinical manifestations include menstrual irregularities, infertility, hirsutism, acne, obesity, insulin resistance, and metabolic syndrome, all of which substantially impair quality of life. Diagnosis is based on established criteria, including the Rotterdam, National Institutes of Health (NIH), and Androgen Excess Society (AES) guidelines, after excluding other causes of hyperandrogenism and ovulatory dysfunction. Management requires an individualized, multidisciplinary approach that integrates lifestyle modification with pharmacological therapy according to patients' reproductive goals and metabolic risk factors. Lifestyle interventions, including dietary modification, regular physical activity, and behavioral support, remain the cornerstone of treatment, while medications such as combined oral contraceptives, metformin, letrozole, clomiphene citrate, and anti-androgens are used to address specific clinical manifestations. Women with PCOS are at increased risk of long-term complications, including type 2 diabetes mellitus, cardiovascular disease, endometrial cancer, pregnancy complications, and mental health disorders, necessitating ongoing monitoring and preventive care. Emerging research into biomarkers, gut microbiota, precision medicine, and genetic and epigenetic mechanisms offers promising opportunities for earlier diagnosis and more personalized therapeutic strategies. Continued research is essential to improve clinical outcomes and reduce the long-term burden of PCOS.

Keywords: Polycystic ovary syndrome (PCOS), hyperandrogenism, insulin resistance, hyperinsulinemia, infertility, hirsutism.

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Introduction

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine and metabolic disorders affecting women of reproductive age, with an estimated global prevalence ranging from 8% to 13%, depending on the diagnostic criteria and population studied (Teede et al., 2023). PCOS is characterized by a combination of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, although not all affected individuals present with the same clinical features. The syndrome is associated with significant reproductive, metabolic, and psychological consequences, including infertility, menstrual irregularities, insulin resistance, obesity, type 2 diabetes mellitus, cardiovascular risk factors, anxiety, and depression (1,2).

The etiology of PCOS is complex and multifactorial, involving interactions between genetic susceptibility, hormonal imbalance, environmental influences, and lifestyle factors. Insulin resistance plays a central role in the pathophysiology of the disorder by exacerbating hyperandrogenism and disrupting normal ovarian function (3). Due to its heterogeneous presentation, the diagnosis of PCOS is commonly based on the Rotterdam criteria, which require the presence of at least two of the following three features after excluding other etiologies: oligo- or anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound (4).

Given its lifelong health implications, early diagnosis and comprehensive management of PCOS are essential. Current evidence emphasizes a multidisciplinary approach that includes lifestyle modification, pharmacological therapy when indicated, and long-term monitoring of metabolic and reproductive health. Continued research is focused on improving diagnostic accuracy, understanding the underlying mechanisms of the syndrome, and developing personalized treatment strategies to optimize patient outcomes (2).

I. Epidemiology and Prevalence

PCOS affects a substantial proportion of women globally, with prevalence estimates varying depending on the diagnostic criteria employed. Recent meta-analyses indicate a global prevalence of approximately 9.2% (95% CI: 6.8–12.5%), though this figure ranges from 5% to 21% across different populations and studies. When stratified by diagnostic criteria, prevalence is 5.5% using NIH criteria, 11.5% using Rotterdam criteria, and 7.1% using AES criteria (5,6).

Among adolescent females aged 10–24 years, PCOS affects 6–18% of this population, highlighting the importance of early recognition and intervention. The global burden of PCOS has increased substantially over recent decades, with age-standardized prevalence rates rising by 28.21% since 1990. In 2021 alone, there were approximately 69.47 million prevalent cases globally, representing an age-standardized rate of 867.7 per 100,000 women. Regional variations exist, with particularly high prevalence noted in African populations, necessitating targeted public health measures for timely prevention and management (6,7).

II. Pathophysiology and Etiology

The pathophysiology of PCOS is multifactorial and heterogeneous, shaped by intricate interactions between genetic, epigenetic, metabolic, and environmental factors. Three principal characteristics define the pathophysiology: an interaction between reproductive dysfunction and metabolic disorders, high familial aggregation and heritability, and substantial contribution from environmental factors (8,9).

1. Functional Ovarian Hyperandrogenism (FOH)

The vast majority of PCOS cases are explicable by functional ovarian hyperandrogenism (FOH), which appears to arise from intraovarian flaws in the

downregulation of androgen secretion in response to luteinizing hormone (LH). Hyperandrogenism represents a core pathophysiological feature, with increased androgen synthesis lying at the center of the disorder (10,11).

2. Insulin Resistance and Hyperinsulinemia

Insulin-resistant hyperinsulinism is present in approximately half of PCOS cases and serves to aggravate ovarian hyperandrogenism. Insulin resistance exacerbates hyperandrogenism through multiple mechanisms, including increased ovarian androgen production and decreased hepatic sex hormone-binding globulin (SHBG) synthesis. The pathophysiology includes intrinsic ovarian dysfunction strongly affected by external factors such as hyperinsulinemia and hypothalamic-pituitary-ovarian axis disorders (9,10).

3. Genetic and Environmental Factors

PCOS is a complex trait resulting from the interaction of multiple heritable and environmental factors, with a "two-hit" hypothesis proposing that PCOS results from a congenitally programmed predisposing factor that becomes clinically manifest upon exposure to postnatal provocative factors. Genetic factors contribute significantly, with high familial aggregation observed, though precise mechanisms underlying heritability remain to be fully determined. Environmental factors may include prenatal exposure to the intrauterine environment of mothers with PCOS, the follicular microenvironment, and postnatal lifestyle factors (9,10).

4. Hypothalamic-Pituitary-Ovarian Axis Dysfunction

Aberrant gonadotropin-releasing hormone (GnRH) pulsation and the resulting abnormal gonadotropin secretion contribute to the pathophysiology, with hyperandrogenism, ovulatory dysfunction, and insulin resistance interacting and exacerbating one another. Disrupted folliculogenesis represents another core pathophysiological feature of the syndrome (11,12).

III. Clinical Features and Symptoms

PCOS presents with a diverse array of clinical manifestations, though hyperandrogenism and anovulation remain the most common symptoms across different phenotypes. The syndrome typically affects multiple organ systems, producing both reproductive and metabolic sequelae (13).

1. Reproductive and Dermatological Features

The major clinical manifestations include:

- **Menstrual irregularities**: Irregular or infrequent menstrual periods, oligomenorrhea, and amenorrhea are hallmark features (13).
- **Hyperandrogenism**: Clinical signs include hirsutism (excess hair growth), acne, and androgenic alopecia (13).
- **Anovulation and infertility**: Chronic anovulation leads to subfertility and represents one of the most common causes of anovulatory infertility (14,15).
- **Polycystic ovaries**: Multiple small follicles visible on ultrasound examination (16).

2. Metabolic Features

- **Obesity and weight gain**: Particularly central adiposity, though PCOS affects women across all body mass indices (16).
- **Insulin resistance**: Present in a substantial proportion of women with PCOS, independent of obesity (16,17).
- **Acanthosis nigricans**: Dark, velvety skin patches indicating insulin resistance (18).

3. Other Clinical Features

Women with PCOS may experience pain (including pelvic pain), mood disturbances, sleep disorders, and reduced quality of life. The heterogeneous presentation necessitates comprehensive evaluation for accurate diagnosis (8).

IV. Diagnosis and Diagnostic Criteria

There is no single diagnostic test for PCOS; instead, diagnosis requires a combination of clinical, biochemical, and imaging findings after exclusion of other causes. Multiple diagnostic criteria exist, contributing to variability in prevalence estimates and clinical practice (13).

1. Rotterdam Criteria (2003)

The most widely used criteria require at least two of the following three features:

1. Oligo-ovulation or anovulation
2. Clinical and/or biochemical signs of hyperandrogenism
3. Polycystic ovaries on ultrasound (≥12 follicles measuring 2–9 mm in diameter and/or increased ovarian volume >10 mL)

Other etiologies of hyperandrogenism and ovulatory dysfunction must be excluded (5, 19).

2. NIH Criteria (1990)

More stringent criteria requiring both:

1. Clinical or biochemical hyperandrogenism
2. Oligo-ovulation or anovulation

With exclusion of other causes (5,20).

3. Androgen Excess Society (AES) Criteria (2006)

Requires:

1. Clinical or biochemical hyperandrogenism
2. Ovarian dysfunction (oligo-anovulation and/or polycystic ovaries)

With exclusion of other androgen excess or related disorders (5).

4. Diagnostic Workup

A comprehensive evaluation includes:

- **Medical history**: Menstrual history, symptoms of hyperandrogenism, weight changes, family history.
- **Physical examination**: Assessment for hirsutism, acne, alopecia, acanthosis nigricans, and BMI calculation.
- **Biochemical tests**: Total and free testosterone, SHBG, LH, FSH, prolactin, TSH, 17-hydroxyprogesterone, and metabolic screening (fasting glucose, lipid profile) (13).
- **Pelvic ultrasound**: To assess ovarian morphology.
- **Exclusion of other conditions**: Thyroid dysfunction, hyperprolactinemia, congenital adrenal hyperplasia, Cushing syndrome, and androgen-secreting tumors (13).

V. Management and Treatment

Management of PCOS is individualized based on patient symptoms, reproductive goals, and metabolic risk profile. Treatment strategies address both short-term symptoms and long-term health risks through a combination of lifestyle interventions, pharmacological therapies, and emerging approaches (21,22).

1. Pharmacological Interventions

A. For Menstrual Regulation and Hyperandrogenism:

- **Combined oral contraceptive pills (OCPs)**: First-line therapy for menstrual irregularity and hyperandrogenism in women not seeking pregnancy.
- **Anti-androgens**: Spironolactone, finasteride, and flutamide for hirsutism and acne, typically combined with OCPs.
- **Topical treatments**: Eflornithine cream for facial hirsutism (22).

B. For Ovulation Induction:

- **Clomiphene citrate**: First-line ovulation induction agent.
- **Letrozole**: Aromatase inhibitor showing superior outcomes in some studies.

- **Metformin**: An insulin sensitizer that may improve ovulation, particularly in insulin-resistant women.
- **Gonadotropins and assisted reproductive technologies**: For clomiphene-resistant cases (21,22).

C. For Metabolic Management:

- **Metformin**: Improves insulin sensitivity, may aid weight management, and reduces diabetes risk (23).
- **GLP-1 receptor agonists**: Emerging evidence for weight loss and metabolic improvement (23).
- **Inositols**: Myo-inositol and D-chiro-inositol showing promise for metabolic and reproductive outcomes (8,21,22).

2. Emerging Therapies

Novel treatments under investigation include microbiota modulation, microRNA therapies, and targeted molecular interventions addressing specific pathophysiological pathways. Comprehensive clinical trials continue to evaluate these approaches (8).

3. Lifestyle Interventions

Lifestyle modification represents the cornerstone and first-line treatment for all women with PCOS, regardless of BMI or reproductive goals. International evidence-based guidelines recommend lifestyle interventions as fundamental to chronic disease prevention and management in PCOS (24,25).

4. Dietary Interventions

Evidence supports various dietary approaches:

- **Caloric restriction**: Weight loss of 5–10% improves reproductive, metabolic, and psychological outcomes.
- **Balanced macronutrient distribution**: No single diet is superior; emphasis on whole foods, reduced processed carbohydrates, and adequate protein.
- **Low glycemic index diets**: May improve insulin sensitivity and metabolic parameters.
- **Mediterranean-style diets**: Associated with improved metabolic profiles and reduced inflammation (26-28).

5. Physical Activity

Exercise provides benefits independent of weight loss:

- **Aerobic exercise**: Improves cardiovascular fitness, insulin sensitivity, and body composition.
- **Resistance training**: Increases lean muscle mass, improves metabolic rate, and enhances insulin sensitivity.
- **Combined interventions**: Diet plus exercise produces superior outcomes compared to either intervention alone, with most studies reporting weight loss ranging from 3% to 10% (15,24,28).

6. Behavioral Interventions

- **Cognitive behavioral therapy**: Addresses emotional eating, body image concerns, and psychological distress.
- **Motivational interviewing**: Enhances adherence to lifestyle changes.
- **Structured programs**: Multicomponent interventions including education, goal-setting, and self-monitoring show greatest efficacy (26,27,29).

Evidence for Lifestyle Interventions

Systematic reviews demonstrate that lifestyle interventions improve free androgen index (FAI), weight, BMI, and reproductive outcomes in women with PCOS. However, robust clinical trials are warranted to expand the evidence base for specific lifestyle components beyond population recommendations for diet and physical activity (26,30).

VI. Long-term Health Complications

PCOS carries significant long-term health implications beyond reproductive concerns, necessitating comprehensive screening and preventive strategies (31,32).

1. Metabolic Complications

- **Type 2 Diabetes Mellitus**: Women with PCOS have a 3–7-fold increased risk of developing type 2 diabetes, independent of obesity (31).
- **Metabolic Syndrome**: Increased prevalence of metabolic syndrome, characterized by central obesity, dyslipidemia, hypertension, and impaired glucose tolerance (33).
- **Dyslipidemia**: Elevated triglycerides, LDL cholesterol, and reduced HDL cholesterol (17,31,34).

2. Cardiovascular Disease

Cross-sectional and longitudinal studies confirm increased cardiovascular risk:

- **Hypertension**: Higher prevalence and earlier onset compared to unaffected women.
- **Subclinical atherosclerosis**: Increased carotid intima-media thickness and coronary artery calcification.
- **Cardiovascular events**: Emerging data suggest increased risk of myocardial infarction and stroke, though long-term prospective data remain limited (32,34,35).

3. Reproductive and Gynecological Complications

- **Endometrial hyperplasia and cancer**: Chronic anovulation and unopposed estrogen exposure increase risk 2–6-fold.
- **Infertility**: PCOS accounts for 75–80% of anovulatory infertility cases.
- **Pregnancy complications**: Increased risk of gestational diabetes, preeclampsia, preterm birth, and miscarriage (31,32).

4. Psychological and Quality of Life

- **Depression and anxiety**: 2–3-fold increased prevalence of mood disorders.
- **Reduced quality of life**: Impaired health-related quality of life across multiple domains.
- **Eating disorders**: Higher prevalence of disordered eating patterns (8,36).

5. Other Long-term Risks

- **Obstructive sleep apnea**: Increased prevalence independent of BMI.
- **Non-alcoholic fatty liver disease (NAFLD)**: Higher risk of hepatic steatosis and fibrosis.
- **Chronic kidney disease**: Emerging evidence suggests increased risk (17,32).

VII. Future Directions and Research

The evolving understanding of PCOS pathophysiology continues to inform novel diagnostic and therapeutic approaches.

1. Biomarkers and Diagnostics

- **Anti-Müllerian Hormone (AMH)**: Elevated AMH levels correlate with polycystic ovarian morphology and may serve as a diagnostic biomarker.
- **MicroRNAs**: Circulating microRNAs show promise as diagnostic and prognostic markers.
- **Metabolomics and proteomics**: Identification of novel biomarkers for early detection and phenotyping (8,21).

2. Gut Microbiota

Emerging evidence implicates gut microbiota dysbiosis in PCOS pathophysiology, with microbiota modulation representing a potential therapeutic target (8).

3. Precision Medicine

Recognition of PCOS heterogeneity has prompted calls for phenotype-specific management strategies,

moving beyond one-size-fits-all approaches to personalized, multidisciplinary care (8).

4. Genetic and Epigenetic Research

Ongoing genome-wide association studies (GWAS) and epigenetic investigations aim to clarify genetic architecture and gene-environment interactions, potentially enabling risk prediction and targeted prevention (9,10).

5. Long-term Outcome Studies

Large-scale prospective studies are needed to better characterize long-term cardiovascular, metabolic, and cancer risks, informing evidence-based screening and prevention guidelines (32,34).

Conclusion

PCOS represents a complex, heterogeneous endocrine and metabolic disorder affecting 6–20% of women of reproductive age worldwide, with rising prevalence and substantial global health burden. The syndrome is characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, arising from intricate interactions among genetic, epigenetic, metabolic, and environmental factors.

Diagnosis requires careful clinical evaluation using established criteria (Rotterdam, NIH, or AES) with exclusion of other causes, while management must be individualized based on patient symptoms, reproductive goals, and metabolic risk profile. Lifestyle interventions remain the cornerstone of therapy, with pharmacological treatments addressing specific symptoms and metabolic abnormalities.

The long-term health implications of PCOS extend far beyond reproductive concerns, encompassing increased risks of type 2 diabetes, cardiovascular disease, endometrial cancer, and psychological morbidity. Comprehensive, multidisciplinary care addressing both immediate symptoms and long-term health risks is essential for optimizing outcomes in women with PCOS.

Future research directions include biomarker discovery, gut microbiota modulation, precision medicine approaches, and large-scale prospective studies to better characterize long-term risks and inform evidence-based prevention strategies. As understanding of PCOS pathophysiology continues to evolve, so too will opportunities for more targeted, effective interventions to improve the lives of millions of women affected by this common yet complex disorder.

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