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Review Article

Polycystic Ovarian Disease (PCOD)

Mansi Kedar*, Pratiksha Wadge, Vedant Mahale, Sidhi Pawar

K.B.H.S.S Trust Institute of Pharmacy, Malegaon, Maharashtra 423105

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ABSTRACT

Polycystic Ovarian Disease (PCOD), often used interchangeably with Polycystic Ovary Syndrome (PCOS), is one of the most prevalent endocrine disorders affecting women of reproductive age worldwide. It is characterized by chronic an ovulation, hyperandrogenism, and polycystic ovarian morphology, and is frequently associated with insulin resistance, obesity, and sub fertility. The etiology of PCOD is multifactorial, involving genetic predisposition, hormonal imbalances, and environmental influences. Insulin resistance plays a central role in its pathophysiology, exacerbating androgen excess and disrupting normal follicular development. Clinically, PCOD manifests as menstrual irregularities, hirsutism, acne, infertility, and metabolic complications such as type 2 diabetes and cardiovascular risk. Diagnosis is primarily based on the Rotterdam criteria, with variations depending on regional and clinical guidelines. Management focuses on lifestyle modification, pharmacological intervention, and in some cases, surgical treatment. Recent advances highlight the role of gut microbiota, epigenetic, and digital health tools in improving diagnosis and care. Despite growing awareness, diagnostic challenges and under-recognition persist, especially in adolescent and lean phenotypes. This review aims to consolidate current evidence on PCOD, emphasizing early detection, individualized therapy, and the importance of multidisciplinary approaches to improve reproductive, metabolic, and psychological outcomes in affected women.

INTRODUCTION

Polycystic Ovarian Disease (PCOD), commonly equated with Polycystic Ovary Syndrome (PCOS), represents the most prevalent endocrine disorder among reproductive-age women globally. Characterized by chronic anovulation, biochemical or clinical hyperandrogenism, and polycystic ovarian morphology on ultrasound, PCOD manifests with profound reproductive, metabolic, and psychological consequences. Prevalence estimates vary widely ranging from approximately 4–8% under NIH/NICHD criteria to as high as 15–20% under Rotterdam criteria reflecting differences in diagnostic thresholds and

***Corresponding Author:** Deore Harshal
Address: *K.B.H.S.S Trust Institute of Pharmacy, Malegaon, Maharashtra 423105*
Email ✉: mansikedar02@gmail.com

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population characteristics. In South Asian contexts such as India, reported rates frequently exceed 10% among young women, likely due to rising obesity, sedentary lifestyles, and nutritional transitions.0

Although the terms PCOD and PCOS are often used interchangeably, there is regional nuance in terminology. In some countries, especially India, the term PCOD is preferred, sometimes implying a relatively milder or transient variant of the syndrome, whereas PCOS is considered to represent more persistent systemic metabolic dysfunction.

3.1 Burden of Disease

The impact of PCOD is multifaceted. Women commonly present with menstrual irregularities such as oligomenorrhea or amenorrhea, alongside clinical signs of hyperandrogenism namely hirsutism, acne, and androgenic alopecia. Infertility and subfertility frequently result from anovulation. On the metabolic front, insulin resistance is a hallmark feature, affecting up to 70% of affected individuals, and contributes to increased risk of type 2 diabetes, dyslipidemia, hypertension, non-alcoholic fatty liver disease (NAFLD), and cardiovascular morbidity. Moreover, women with PCOD face significantly elevated risks of endometrial hyperplasia and carcinoma, particularly in the context of chronic unopposed estrogen exposure.

Equally important is the psychological burden: depression, anxiety, body image concerns, and decreased quality of life are highly prevalent, often exacerbated by delayed diagnosis and suboptimal support.

3.2 Etiological Underpinnings

The etiology of PCOD is complex and multifactorial, encompassing genetic, epigenetic, hormonal, metabolic, and environmental factors. Family clustering, high concordance in monozygotic twins compared to dizygotic twins, and identification of susceptibility loci such as DENND1A and THADA in genome-wide association studies underscore significant heritability. Additionally, prenatal androgen exposure and epigenetic modifications may influence susceptibility from early developmental stages.

Central to pathophysiology is insulin resistance, which amplifies ovarian androgen production and disrupts follicular development. This overt hyperinsulinemia stimulates LH secretion, interferes with SHBG synthesis, and contributes to elevated free androgens, reinforcing the cycle of metabolic and reproductive dysfunction .

HPO axis dysregulation results in persistently elevated LH-to-FSH ratios, defective steroidogenesis, and arrested follicle maturation leading to characteristic polycystic ovarian morphology .

Emerging data implicate chronic low-grade inflammation and oxidative stress as contributors to PCOD pathology. Elevated markers of inflammation including IL-6, TNF-α, IL-18, and CRP are consistently observed in PCOD cohorts, even independent of obesity. Hyperhomocysteinemia has also been associated with impaired ovulation and metabolic disruption in these women .

3.3 Phenotypic Heterogeneity

Under the Rotterdam criteria, PCOD is phenotypically classified into four groups based on presence or absence of hyperandrogenism (HA),

ovulatory dysfunction (OD), and polycystic ovarian morphology (PCOM):

- Phenotype A: HA + OD + PCOM (most prevalent and metabolically severe)
- B: HA + OD
- C: HA + PCOM
- D: OD + PCOM (mildest phenotype).

These phenotypes differ substantially in their severity of metabolic and reproductive risk, with phenotype A associated with the highest risk for insulin resistance, dyslipidemia, and clomiphene resistance .

3.4 Diagnosis

Diagnostic frameworks have evolved over decades. The NIH (1990) criteria require both ovulatory dysfunction and clinical/biochemical hyperandrogenism. The Rotterdam consensus (2003) broadened the criteria to include PCOM, requiring any two of the three features leading to expanded prevalence estimates. The AE-PCOS Society criteria (2006) emphasize hyperandrogenism plus one of the other features, reflecting differences in sensitivity and specificity across criteria sets .

Updated 2023 international guidelines now consider anti-Müllerian hormone (AMH) as a surrogate for ultrasound in adults and adolescents when appropriate, thereby streamlining diagnosis in resource-limited settings. Importantly, diagnostic exclusion of other endocrinopathies such as thyroid disease, hyperprolactinemia, and non-classic congenital adrenal hyperplasia is central to accurate identification.

3.5 Purpose of Review

Despite intensive research, challenges persist in early detection and effective management, especially among adolescents, lean phenotypes, and underserved populations. Misunderstanding of terminology, disparities in care, and the burden of misinformation complicate patient experiences.

This review aims to synthesize up-to-date epidemiological evidence, elucidate underlying mechanisms, clarify diagnostic frameworks, compare clinical phenotypes, and evaluate both established and emerging therapeutic strategies. By integrating recent insights from gut microbiota and genetic studies to AI-assisted diagnostics this paper seeks to support early recognition, personalized management, and multidisciplinary strategies to improve reproductive, metabolic, and psychological outcomes for women affected by PCOD.

4. Epidemiology of PCOD/PCOS

4.1 Global Prevalence and Regional Statistics

Meta-analyses estimate that approximately 9.2% (95% CI: 6.8–12.5%) of women of reproductive age worldwide have PCOS, with variations across diagnostic criteria: 5.5% using NIH criteria, 11.5% via Rotterdam, and 7.1% with AES standards .According to the Global Burden of Disease 2021 data, about 65.8 million women aged 15–49 globally lived with PCOS in 2021; both incidence and DALYs have increased by nearly 50% to 90% since 1990. Age-standardized prevalence reached ~867.7 per 100,000 (≈0.87%) in 2021, up ~28% .

Regionally, high SDI (Socio-demographic Index) areas (e.g., high-income Asia Pacific, Europe) show the highest documented prevalence (~225 to ~838 per 100,000), while low-SDI regions report lower registered rates often due to underdiagnosis or limited healthcare infrastructure.

South Asia leads in absolute numbers reporting over 112.9 million prevalent cases and ~416,000 incident cases in 2021. India accounted for one of the highest national burdens (~347,000 cases in 2021) .

4.2 Age Groups Affected

PCOS commonly begins in adolescence or early adulthood, with peak incidence between ages 15–19, followed by sustained elevated prevalence in women aged 20–49.

The highest rise in PCOS burden has been observed in the 45–49 age group, particularly in middle-SDI regions, where disease burden has surged over the last three decades.

Adolescents remain a key demographic: about 90% of cases in both 1990 and 2021 occurred in the 10–19 age bracket.

4.3 Rising Trends due to Lifestyle & Environmental Factors

Since 1990, global incidence of PCOS increased by ~49%, while prevalence and DALYs rose by ~89% and ~87%, respectively.

The adoption of broader diagnostic criteria (e.g., Rotterdam), improved imaging access, and increased disease awareness have significantly contributed to higher detected prevalence—particularly in high-SDI countries.

Growing rates of obesity and sedentary behaviors are key contributors: obese women have two to three times higher PCOS risk compared to non-obese counterparts.

Exposure to environmental endocrine-disrupting chemicals such as phthalates, PFAS, and heavy metals is increasingly implicated in increased PCOS risk and earlier onset .

Rapid urbanization and lifestyle changes especially in India have been associated with surging PCOS prevalence. These include dietary shifts, Westernization, altered circadian rhythms, and stress associated with socio-economic transitions.

Summary Table

Aspect	Key Points
Global Prevalence	~9.2% overall; varies by criteria (NIH ~5.5%, Rotterdam ~11.5%)
Rising Burden	Incidence ↑ ~49%, prevalence ↑ ~28–89% since 1990
Age Groups	Highest in adolescent age (15–19); significant rise in 45–49 group
Regional Variability	High SDI regions report higher rates; South Asia highest in absolute cases
Contributing Factors	Obesity, sedentary lifestyle, endocrine disruptors, improved diagnosis

5. Etiology & Risk Factors of PCOD/PCOS

5.1 Genetic Factors

Genetics play a significant role in the predisposition to PCOD, with strong evidence of familial clustering. Women with a family history of PCOS are at higher risk, suggesting a heritable component. Genome-wide association studies (GWAS) have identified multiple susceptibility loci linked to the syndrome, including polymorphisms in genes such as DENND1A, THADA, FTO, and LHCGR. These genes influence ovarian function, steroidogenesis, and metabolic regulation. However, the inheritance pattern is complex and polygenic, involving interactions with environmental factors, which may explain variability in phenotypes and severity across individuals .

5.2 Hormonal Imbalances

The hallmark hormonal disturbance in PCOD is hyperandrogenism, characterized by elevated serum androgens such as testosterone, androstenedione, and dehydroepiandrosterone sulfate (DHEAS). Increased ovarian androgen production disrupts normal follicular development, leading to anovulation. Another key hormonal imbalance is the altered luteinizing hormone (LH) to follicle-stimulating hormone (FSH) ratio, often elevated (>2:1), which promotes excess androgen synthesis by ovarian theca cells. This dysregulated hypothalamic-pituitary-ovarian (HPO) axis contributes to menstrual irregularities and polycystic ovarian morphology

5.3 Insulin Resistance Central Role

Insulin resistance (IR) is considered a central pathogenic factor in PCOD, affecting up to 70% of patients regardless of obesity status. Compensatory hyperinsulinemia enhances ovarian androgen production by acting synergistically with LH on theca cells, decreases hepatic synthesis of sex hormone-binding globulin (SHBG), resulting in higher free androgen levels, and exacerbates metabolic dysfunction. IR also contributes to impaired glucose tolerance and increases the risk of type 2 diabetes and cardiovascular disease in PCOD patients. The precise molecular mechanisms linking IR and PCOD involve defects in insulin receptor signaling pathways and post-receptor mechanisms .

5.4 Lifestyle and Obesity

Obesity significantly worsens the clinical and metabolic manifestations of PCOD. Adiposity increases peripheral conversion of androgens, exacerbates insulin resistance, and alters adipokine secretion, which collectively contribute to the syndrome's severity. Poor dietary habits characterized by high glycemic index foods, saturated fats, and low fiber intake along with sedentary lifestyles, are major modifiable risk factors. Weight gain and physical inactivity fuel insulin resistance and hormonal disturbances, establishing a vicious cycle. Lifestyle interventions focusing on diet and exercise remain the cornerstone of management due to their positive impact on hormonal balance and metabolic outcomes .

5.5 Environmental Factors

Emerging research highlights the role of endocrine-disrupting chemicals (EDCs) such as phthalates, bisphenol A (BPA), perfluoroalkyl substances (PFAS), and heavy metals in increasing PCOD risk. These compounds can interfere with hormone synthesis, metabolism, and receptor signaling, potentially triggering or worsening PCOD features. Chronic exposure to EDCs may contribute to early-onset PCOD and metabolic disturbances by altering the HPO axis and insulin sensitivity. Urbanization and industrialization have led to increased EDC exposure, underscoring the importance of environmental health in PCOD pathogenesis.

Summary Table

Etiological Factor	Description
Genetic Factors	Family history, gene polymorphisms (DENND1A, THADA, etc.); complex polygenic inheritance
Hormonal Imbalances	Elevated androgens; increased LH/FSH ratio; HPO axis dysregulation
Insulin Resistance	Central driver; hyperinsulinemia increases ovarian androgen synthesis; worsens metabolic profile
Lifestyle & Obesity	Poor diet, sedentary behavior; obesity amplifies hormonal and metabolic abnormalities
Environmental Factors	Endocrine disruptors (phthalates, BPA, PFAS) affect hormone pathways and insulin sensitivity

6. Pathophysiology of PCOD/PCOS

6.1 Hormonal Mechanisms

PCOD is characterized primarily by hormonal imbalances that disrupt normal ovarian function. Central to its pathophysiology is hyperandrogenism, resulting from increased production of androgens by ovarian theca cells. Excess luteinizing hormone (LH) secretion stimulates theca cells to synthesize androgens such as testosterone and androstenedione. At the same time, decreased levels or relative insufficiency of follicle-stimulating hormone (FSH) impair granulosa cell function and estrogen synthesis, disrupting follicle maturation. Moreover, reduced production of sex hormone-binding globulin (SHBG), often due to hyperinsulinemia, increases the bioavailability of free androgens, exacerbating clinical symptoms such as hirsutism, acne, and alopecia .

6.2 Ovarian Dysfunction and Follicular Arrest

A hallmark of PCOD is the arrest of follicular development at the small antral stage. Normally, a dominant follicle emerges during the menstrual cycle to ovulate, but in PCOD, multiple small follicles accumulate, leading to the characteristic polycystic ovarian morphology visible on ultrasound. This follicular arrest is partly due to hormonal imbalances but also to intrinsic ovarian factors, including altered local growth factors and disrupted intraovarian paracrine signaling. The failure of follicles to mature prevents ovulation, resulting in chronic anovulation and irregular menstruation.

6.3 Role of Insulin Resistance in Exacerbating Hyperandrogenism

Insulin resistance is pivotal in the pathogenesis of PCOD, occurring in both obese and lean patients. Insulin acts synergistically with LH to stimulate androgen production in ovarian theca cells, intensifying hyperandrogenism. Furthermore, hyperinsulinemia suppresses hepatic synthesis of SHBG, increasing circulating free androgen levels. Elevated insulin levels also disrupt follicular development by affecting granulosa cell function. Collectively, insulin resistance links metabolic dysfunction with reproductive abnormalities in PCOD, contributing to increased risks of type 2 diabetes, cardiovascular disease, and infertility .

6.4 Hypothalamic–Pituitary–Ovarian (HPO) Axis Disruption

PCOD involves dysregulation of the HPO axis, resulting in abnormal gonadotropin secretion. Increased frequency and amplitude of gonadotropin-releasing hormone (GnRH) pulses favor LH secretion over FSH, leading to an elevated LH/FSH ratio typical of PCOD. This imbalance promotes androgen excess and impairs follicular maturation. Moreover, altered feedback sensitivity to estrogen and progesterone disrupts the cyclical regulation of GnRH and gonadotropins. Neuroendocrine abnormalities, including altered kisspeptin and GABAergic signaling, may further contribute to this dysregulation. The disturbed HPO axis thus plays a crucial role in the hormonal and ovulatory disturbances observed in PCOD ..

7. Clinical Manifestations of PCOD/PCOS

PCOD presents with a diverse range of symptoms, reflecting its complex hormonal and metabolic disruptions.

7.1 Menstrual Irregularities

The most common clinical sign is menstrual dysfunction, including oligomenorrhea

(infrequent menstrual periods) and amenorrhea (absence of menstruation). These irregularities result from chronic anovulation caused by disrupted follicular development and hormonal imbalances. Women with PCOD often experience unpredictable or absent cycles, leading to difficulties in conceiving and increased risk of endometrial hyperplasia.

7.2 Androgenic Symptoms

Elevated androgen levels lead to classic hyperandrogenic features such as acne, hirsutism (excessive terminal hair growth in a male-pattern distribution), and androgenic alopecia (hair thinning or male-pattern baldness). These symptoms can cause significant distress and affect self-esteem, particularly in adolescents and young women.

7.3 Infertility and Subfertility

PCOD is a leading cause of infertility due to chronic anovulation. The failure of follicular maturation and ovulation prevents regular conception. However, many women can conceive with medical assistance such as ovulation induction therapies.

7.4 Obesity and Insulin Resistance

A significant proportion of PCOD patients exhibit obesity, especially central adiposity, which exacerbates insulin resistance. Insulin resistance worsens hyperandrogenism and increases the risk of metabolic complications like type 2 diabetes and cardiovascular disease. Even lean women with PCOD may have underlying insulin resistance.

7.5 Psychological Symptoms

PCOD often negatively impacts mental health, with increased prevalence of anxiety, depression, and poor body image. The combination of physical symptoms, fertility challenges, and social stigma contributes to psychological distress, emphasizing the need for holistic management addressing both physical and mental health.

7.6 Diagnostic Criteria for PCOD/PCOS

7.6.1 Rotterdam Criteria (2003)

The most widely accepted criteria require the presence of any two of the following three features:

Oligo- or anovulation (irregular or absent menstrual cycles)

Clinical or biochemical signs of hyperandrogenism (e.g., hirsutism, elevated testosterone)

Polycystic ovarian morphology on ultrasound (≥12 follicles in one ovary or increased ovarian volume >10 cm^3)

7.6.2 NIH Criteria (1990)

Defines PCOS as both hyperandrogenism and chronic anovulation, excluding other causes.

7.6.3 AE-PCOS Society Criteria (2006)

Focuses on hyperandrogenism as a mandatory criterion plus either ovulatory dysfunction or polycystic ovaries.

7.7 Diagnostic Investigations

7.7.1 Ultrasound: Transvaginal ultrasound reveals ≥12 small follicles (2–9 mm) arranged peripherally ("string of pearls") and/or increased ovarian volume.

7.7.2 Blood Tests: Measure LH, FSH (often elevated LH/FSH ratio), serum testosterone (total and free), anti-Müllerian hormone (AMH)

(elevated in PCOD), fasting insulin, and glucose levels to assess metabolic status.

7.7.3 Exclusion of Other Disorders: Essential to rule out thyroid dysfunction, hyperprolactinemia, congenital adrenal hyperplasia, and androgen-secreting tumors through appropriate hormonal assays and clinical evaluation.

8. Difference Between PCOD and PCOS

- PCOD (Polycystic Ovarian Disease) is often considered a milder, sometimes temporary ovarian condition characterized mainly by the presence of multiple cysts on the ovaries and irregular menstruation.
- PCOS (Polycystic Ovary Syndrome) is a broader, systemic endocrine disorder involving hormonal imbalance, metabolic issues, and chronic symptoms affecting multiple body systems.
- PCOD symptoms may resolve with lifestyle changes or treatment, while PCOS tends to be a chronic, lifelong condition requiring ongoing management.
- PCOS is associated with more pronounced insulin resistance, higher risk of metabolic syndrome, and greater infertility challenges.
- PCOD is sometimes used interchangeably with PCOS but lacks the systemic metabolic and hormonal complexities seen in PCOS.

Summary Table

Feature	PCOD	PCOS
Definition	Ovarian condition with multiple cysts	Endocrine disorder with systemic effects
Severity	Generally milder, sometimes reversible	More severe and chronic
Hormonal Imbalance	Less pronounced	Significant hyperandrogenism and LH/FSH ratio changes
Metabolic Issues	Usually absent or mild	Common, including insulin resistance and obesity
Symptoms	Mostly ovarian cysts and menstrual irregularities	Includes cysts, hirsutism, obesity, infertility, metabolic syndrome
Long-Term Risks	Lower risk	Higher risk of diabetes, cardiovascular disease, endometrial cancer
Treatment	Often lifestyle changes and symptomatic care	Requires comprehensive hormonal, metabolic, and lifestyle management

9. Management Strategies for PCOD/PCOS

9.1 Lifestyle Modifications (First-Line)

Lifestyle changes form the cornerstone of PCOD management, especially in overweight or obese patients.

- **9.1.1 Diet:** Emphasis on low glycemic index (GI) foods and Mediterranean-style diets rich in whole grains, fruits, vegetables, healthy fats, and lean proteins helps improve insulin sensitivity and hormonal balance.
- **9.1.2 Exercise:** Regular physical activity enhances insulin action, promotes weight loss, and improves menstrual regularity and ovulation. Weight management through combined diet and exercise reduces metabolic and reproductive complications.

9.2 Pharmacological Treatments

When lifestyle modifications are insufficient, medications are used to address hormonal and metabolic disturbances:

- **Metformin:** An insulin sensitizer that improves insulin resistance, lowers androgen levels, and can restore ovulation.
- **Oral Contraceptive Pills (OCPs):** Regulate menstrual cycles, reduce androgen levels, and improve symptoms like hirsutism and acne.
- **Anti-Androgens:** Agents like spironolactone block androgen receptors, helping reduce hair growth and acne.
- **Ovulation Inducers:** Clomiphene citrate and letrozole are used to stimulate ovulation in women seeking pregnancy.

9.3 Surgical Intervention

- **Laparoscopic Ovarian Drilling (LOD):** A minimally invasive surgical option for clomiphene-resistant infertility. It reduces ovarian androgen production and can restore ovulation but is considered a last resort due to potential risks.

9.4 Complementary/Alternative Medicine

- **Ayurveda and Homeopathy:** Widely used in some regions but require cautious mention due to limited high-quality evidence.
- **Yoga and Mindfulness:** Growing evidence supports their role in stress reduction, improving psychological well-being, and possibly aiding metabolic health.

10. Recent Advances & Future Directions in PCOD/PCOS

10.1 Role of Gut Microbiota

Emerging research highlights the gut microbiome's influence on metabolic and inflammatory pathways in PCOD. Dysbiosis (imbalanced gut flora) may exacerbate insulin resistance and hormonal disturbances, suggesting probiotics and microbiota-targeted therapies as promising interventions.

10.2 Genomics and Epigenetics

Advances in genetic studies have identified multiple gene polymorphisms linked to PCOD susceptibility. Epigenetic modifications, including DNA methylation and histone changes, are being explored for their roles in gene-environment interactions affecting disease expression, paving the way for more precise diagnostics and treatments.

10.3 Personalized Medicine and Targeted Therapies

Tailoring treatment based on individual genetic, metabolic, and phenotypic profiles aims to improve efficacy and reduce side effects. Targeted therapies addressing specific molecular pathways are under investigation, moving beyond one-size-fits-all approaches.

10.4 Artificial Intelligence (AI)

AI applications in PCOD include enhanced ultrasound image analysis for improved detection of polycystic ovaries and AI-driven algorithms for patient monitoring and management. These tools can facilitate earlier diagnosis, personalized treatment plans, and better outcome tracking.

10.5 Emerging Nutraceuticals and Natural Supplements

Natural compounds such as inositols, omega-3 fatty acids, and vitamin D are gaining attention for their insulin-sensitizing and anti-inflammatory effects, offering complementary options alongside conventional treatments.

10.6 Telemedicine and Digital Health

The rise of telemedicine and smartphone apps enables remote monitoring of menstrual cycles, symptoms, and treatment adherence. Digital platforms provide education, support, and timely interventions, particularly valuable for patients in underserved areas.

CONCLUSION

Polycystic Ovarian Disease (PCOD) remains one of the most prevalent endocrine disorders affecting women of reproductive age worldwide. This review highlights the multifaceted nature of PCOD, encompassing complex hormonal imbalances, ovarian dysfunction, metabolic disturbances, and significant psychological impacts. The pathophysiology revolves primarily around hyperandrogenism, insulin resistance, and hypothalamic–pituitary–ovarian axis disruption, which collectively contribute to the diverse clinical manifestations such as menstrual irregularities, infertility, androgenic symptoms, obesity, and mental health challenges.

Early detection of PCOD is crucial, given its association with long-term complications including type 2 diabetes, cardiovascular disease, and endometrial cancer. Diagnostic criteria like the Rotterdam guidelines provide a comprehensive framework for identifying the condition by combining clinical, biochemical, and imaging findings. Effective management requires a holistic and individualized approach beginning with lifestyle modifications focusing on diet and physical activity, which remain the cornerstone of treatment. Pharmacological options, surgical interventions, and complementary therapies further assist in managing symptoms and improving quality of life.

Recent advances in understanding the role of gut microbiota, genetics, and epigenetics offer promising avenues for personalized medicine, aiming to tailor treatments based on individual patient profiles. Moreover, the integration of artificial intelligence in diagnostic imaging and digital health technologies like telemedicine and menstrual tracking apps enhances patient monitoring and accessibility to care, especially in underserved regions.

Despite these advances, significant gaps remain in our knowledge, particularly concerning the condition's heterogeneity across different ethnic and socio-economic populations. Many studies have predominantly focused on limited cohorts, highlighting the urgent need for larger, diverse population-based research to better understand the variations in presentation, progression, and response to therapy.

In conclusion, PCOD is a complex disorder requiring early recognition and comprehensive management that addresses both the physical and psychological aspects. Multidisciplinary care involving endocrinologists, gynecologists, nutritionists, and mental health professionals is vital for optimal outcomes. Future research should prioritize personalized approaches and equitable healthcare delivery to improve the lives of millions affected by this condition worldwide. With ongoing scientific advancements and increased awareness, there is hope for more effective prevention and treatment strategies in the near future.

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