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Polycystic Ovarian Disease (PCOD) And Women's Health: Challenges And Emerging Solutions

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Abstract
Polycystic ovarian syndrome (PCOS), also known as PCOD, is the most common endocrine condition affecting people with reproductive-age ovaries. It has diverse reproductive, metabolic, and psychological characteristics and poses significant long-term cardiometabolic and reproductive concerns. Prevalence estimates differ depending on diagnostic criteria and geography, however recent meta-analyses and global burden studies put the affected population in the tens of millions globally. Lifestyle modifications, combined oral contraceptives, metformin, and ovulation induction drugs (particularly letrozole) are common treatments, although many patients continue to endure symptoms, comorbidities, and gaps in care. Emerging treatments and techniques (GLP-1 receptor agonists, precision phenotyping, digital health, and integrated models of care) provide promise for improved outcomes, notably in terms of metabolic characteristics and weight management. This article reviews current data on epidemiology, pathophysiology, diagnosis, clinical consequences, current treatments, and possible improvements, as well as identifies gaps and research objectives to improve women's health in the context of PCOS.
Keywords: *PCOS, PCOD, polycystic ovary syndrome, women's health, letrozole, GLP-1 receptor agonists, metabolic risk, infertility, guideline*

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is a complicated, multifaceted disorder that includes reproductive (oligo/anovulation, hyperandrogenism), metabolic (insulin resistance, obesity), and psychological (depression, anxiety) components. Because of its multisystemic character, PCOS is a serious public-health concern: it has an impact on fertility, quality of life, cardiometabolic health, and lifetime illness risk. It is defined by oligo or anovulation, hyperandrogenism, and polycystic ovarian abnormalities. Not only does this cause female infertility, but it also has an impact on the patient's metabolic health.[1] Furthermore, with the acceptance of the "Developmental Origins of Health and Disease" (DOHaD) theory, the focus on the origin of adult disorders has turned to gametogenesis and embryonic development. PCOS moms' metabolic disturbances before to conception increase the likelihood of PCOS in their offspring, resulting in a "vicious cycle". However, due to its high clinical variability, the pathophysiology of PCOS remains unknown, posing significant hurdles for doctors in terms of accurate diagnosis and therapy.[2]

According to estimations from the World Health Organization (WHO), 116 million women worldwide (3.4%) suffered from PCOS in 2012.[3] This high prevalence highlights the substantial financial cost of PCOS, as does its association with irregular menstruation and ovulation, infertility, hair loss, and metabolic problems. While PCOS can develop at any age, starting with menarche, most cases are seen in people between the ages of 20 and 30[4]. 0.43 million disability-adjusted life years (DALYs) are caused by PCOS, which affects 1.55 million women of reproductive age globally. In women of reproductive age, the age-standardized incidence rate of PCOS was 82.44 per 100,000 in 2017, which was 1.45% greater than in 2007. [5]. Although PCOS was once believed to be a condition that only affected adult women, recent research shows that it is actually a lifelong syndrome that initially appears during pregnancy. A mix of environmental and genetic variables is believed to be the main cause of this multifactorial condition, while the precise reason is unknown. Hormonal imbalance, chronic low-grade inflammation, insulin resistance, and hyperandrogenism are the main pathophysiological factors of PCOS. These factors hinder folliculogenesis and raise the risk of associated comorbidities such type II diabetes and endometrial cancer. International guidelines state that ovarian morphology, anovulation, and hyperandrogenism are the three primary criteria used to diagnose PCOS [6].

With a focus on integrated care and translational improvements, this review outlines the state of the art regarding prevalence and burden, pathogenesis, diagnostic techniques, clinical outcomes, proven treatments, and new developments.

Definition

The misconception is mostly caused by the term polycystic, which describes one of the syndrome's impacts on the gonads' radiological appearance, where follicles are halted at different maturational stages but are not actually cysts[7]. The fact that the illness is a metabolic endocrinopathy with consequences that go beyond reproduction is obscured by this nomenclature [8]. The presence of both clinical and biochemical ovulatory failure and high androgen production are the criteria that describe the illness, which is today known as typical PCOS [9].

In an effort to come to an agreement on a broader definition that would encompass women with polycystic ovarian morphology (POM) and ovulatory dysfunction without hyperandrogenism, the American Society of Reproductive Medicine and the European Society of Human Reproduction released the Rotterdam criteria in 2003 [10].

Epidemiology and global burden

Diagnostic criteria have a significant influence on prevalence estimates (NIH, Rotterdam, Androgen Excess Society). Global point prevalence, according to recent meta-analyses, varies widely; pooled estimates range from about 5% to 12%, depending on the criteria, with many studies clustering around 8–10% in populations of reproductive age.[11] According to large burden studies, tens of millions of people are impacted globally (latest estimates in the literature indicate that between 65 and 70 million people will be impacted in 2021). Population growth and increased awareness are probably the main causes of the rising absolute numbers. There are also significant financial and medical expenses associated with the illness. [12]

Etiology:

a. Family Aggregation

There is evidence of family aggregation in the families of women with PCOS, suggesting a possible genetic basis for the etiopathogenesis of the disorder. The interaction of environment and genetics may result in variation in the phenotypic expression of the disease. The search for the genes responsible for polycystic ovarian syndrome has been unsuccessful since only a limited number of genetic differences have been replicated in individuals with the illness from different populations. A potential genetic foundation for the etiopathogenesis of PCOS is suggested by evidence of family aggregation in the families of women with the condition. Variations in the disease's phenotypic expression may arise from the interplay between genetics and environment. Since just a small number of genetic variations have been reproduced in polycystic ovarian syndrome patients from various populations, efforts to identify the genes causing the condition have been fruitless [13].

b. Association between High Testosterone and Insulin Resistance

Patients with normal body weight who are hyperandrogenic have lower insulin sensitivity. Compared to women in good health, this is particularly true for women with PCOS. There is a reciprocal relationship between compensatory hyperinsulinemia with hyperandrogenism and insulin resistance[14]. On the one hand, insulin stimulates the luteinizing hormone (LH), which leads to hyperandrogenemia. A combination of hyperandrogenemia and hyperinsulinemia causes altered LH pulsatility in approximately 50% of women with PCOS. As evidenced by the significant percentage of women with type 1 diabetes, these negative effects of endogenous hyperinsulinism can also occur in exogenous hyperinsulinism situations. [15].

c. Polycystic Ovary Syndrome and Its Heterogeneity

Taking everything into account, abdominal obesity's outward manifestation and the degree of insulin resistance will impact PCOS's phenotypic expression. Based on the current scientific evidence, we believe that PCOS arises from a combination of factors, surplus weight and excessive androgen. This theory states that signs of androgen excess can appear even in weak females if the intrinsic defect is severe enough. On the other hand, a small defect in the process of conversion of cholesterol into steroid hormones will only show clinical symptoms when obesity or insulin resistance [16].

Pathophysiology — an integrated model

PCOS is polygenic and multifactorial. Core pathophysiologic features include:

- **Hypothalamic–pituitary–ovarian axis perturbation:** increased LH pulsatility in many patients, with relative FSH suppression, contributes to ovarian androgen production and follicular arrest.[17]
- **Ovarian dysfunction:** theca cell hyperactivity increases androgen synthesis; folliculogenesis is disrupted leading to anovulation. [18]
- **Insulin resistance & hyperinsulinemia:** central to many PCOS phenotypes; insulin amplifies ovarian androgen production and reduces sex hormone-binding globulin (SHBG), increasing free androgens.[19]

• **Adipose tissue dysfunction & inflammation:** central adiposity and adipokine alterations worsen metabolic and reproductive features.
• **Genetic and developmental factors:** family aggregation and genome-wide studies implicate multiple loci; exposures across the life course (in utero, puberty, lifestyle) modify phenotype expression.

Clinical features and diagnosis

Typical features: menstrual irregularities (oligo/amenorrhea), clinical or biochemical hyperandrogenism (hirsutism, acne, elevated androgens), polycystic ovarian morphology on ultrasound, infertility, obesity, and metabolic dysfunction. [20]

Diagnostic frameworks: The most widely used is the Rotterdam criteria (2003) -diagnosis requires ≥2 of: (1) oligo/anovulation; (2) clinical/biochemical hyperandrogenism; (3) polycystic ovarian morphology — after exclusion of other causes. NIH (1990) criteria are stricter (hyperandrogenism + ovulatory dysfunction). International evidence-based guidelines (2018, updated subsequently) give detailed recommendations on assessment (including biochemical tests, metabolic screening) and management, emphasizing patient-centered care and minimizing unnecessary ovarian imaging in some settings. [21]

Laboratory Evaluation

1) Testosterone

Given the issues with many tests used to measure free testosterone, a total testosterone measurement is likely more accurate than a free testosterone measurement. In PCOS, testosterone levels could be normal. Most testosterone levels in PCOS will be less than 150 ng/dL [22].

2) Dehydroepiandrosterone-sulfate (DHEA-S)

In PCOS, DHEA-S readings can be slightly increased or normal. When DHEA-S levels exceed 800 μg/dL, an adrenal tumor should be taken into account [23].

3) Prolactin

According to reports, 5% to 30% of PCOS patients have mild hyperprolactinemia. Generally speaking, prolactin levels are only 50% higher than the upper bound of normal. It should be noted that hyperprolactinemia is typically a temporary condition, with approximately 3% to 7% of hyperprolactinemic PCOS patients exhibiting consistently increased prolactin levels [24]. This has led to the current belief that hyperprolactinemia and PCOS are separate conditions. Further investigation for additional causes should be conducted if normalization upon resampling does not occur. Polycystic ovaries may be observed on ultrasound in patients with prolactinomas [25].

4) Luteinizing Hormone/Follicle-Stimulating Hormone Ratio

Although not very sensitive or specific, a ratio greater than 2.0 is suggestive of PCOS. Oral contraceptives have an impact on gonadotropin levels [26].

5) Pelvic-ultrasonography

Pelvic ultrasonography can also be very beneficial in the evaluation process; however, over 20% of normal women also have polycystic ovaries, suggesting that the condition is not exclusive to PCOS. In the ultrasound evaluation, the quantity of follicles and ovarian volume are both significant [27].

Health consequences and comorbidities

Short- and long-term consequences include:

• **Infertility and pregnancy complications:** subfertility due to anovulation; higher risks of gestational diabetes and hypertensive disorders.
• **Metabolic disease:** insulin resistance, dyslipidemia, type 2 diabetes (higher lifetime risk), and increased cardiovascular risk markers.
• **Endometrial pathology:** chronic anovulation leads to risk of endometrial hyperplasia and, rarely, cancer.
• **Psychological burden:** elevated rates of depression, anxiety, body-image disturbance, and reduced quality of life.
• **Socioeconomic impacts:** work productivity loss, healthcare costs, and psychosocial stigma.

These wide-ranging effects underscore the need for comprehensive and long-term care pathways. [28]

Established management strategies

to improve fertility in women who want children, avoid endometrial hyperplasia in women with severe ovulatory failure, prevent or treat metabolic problems, and reduce the psycho-emotional impact of acute symptoms of androgen excess. In order to address or avoid smoking, obesity, and sedentary behavior, hygienic-dietetic therapy are typically advised for all patients.[29] Patients with moderate problems might

simply need clinical monitoring to make sure they have more than four to six menstrual cycles each year, which effectively protects the endometrium. Additional tactics are-

Lifestyle first

The cornerstone for PCOS individuals who are overweight or obese is lifestyle modification, which includes food adjustments, increased physical activity, and behavioral support. Metabolic indicators are improved, ovulation is restored in some, and hyperandrogenic symptoms are lessened with even modest weight loss (5–10%).

Pharmacologic treatments for non-fertility goals

- **Combined oral contraceptives (COCs):** first-line treatment for hyperandrogenic symptoms and menstrual control; selection is based on personal risk factors and side effect profile.
- **Metformin:** utilized mostly for insulin resistance and metabolic characteristics; it may also be added for metabolic benefits and in certain fertility regimens.
- **Anti-androgens (spironolactone, cyproterone acetate [region-dependent]):** for hirsutism/acne when contraception is ensured.

Ovulation induction and infertility management

- **Letrozole:** Letrozole, an aromatase inhibitor, is increasingly advised as the first-line pharmacologic ovulation induction since it has demonstrated better ovulation and live-birth rates than clomiphene citrate in numerous randomized trials and meta-analyses in PCOS populations. For resistant patients, combination or sequential methods (letrozole → gonadotropins) are employed.[30]

Obesity and Metabolic Complication

For all PCOS patients, food and hygiene recommendations should be the first line of treatment. Reducing excess weight or maintaining a healthy weight is the goal in order to improve insulin resistance, excess testosterone levels, and body fat distribution. But maintaining lifestyle changes over time can be difficult, especially for those who are moderately to severely obese. [31]

Assisted reproductive technologies & OHSS prevention

The risk of ovarian hyperstimulation syndrome (OHSS) in PCOS patients is reduced by certain stimulation protocols and techniques for IVF, such as low-dose stimulation, GnRH antagonist protocols, use of GnRH agonist trigger, and embryo freezing. In some regimens, metformin as an adjuvant can lower the risk of OHSS.

Emerging solutions & innovations

GLP-1 receptor agonists and other weight-loss agents

Liraglutide, semaglutide, and other GLP-1 receptor agonists exhibit strong weight loss and positive metabolic effects. Although gastrointestinal side effects are common and more research is needed to determine long-term safety data in women of reproductive age, recent systematic reviews and studies in PCOS populations demonstrate significant decreases in BMI, waist circumference, and certain androgenic and metabolic indicators. For obese PCOS patients, GLP-1 RAs show promise, especially if weight loss is the main therapy objective. There are active registries and clinical trials.

Precision phenotyping and biomarkers

In order to move beyond one-size-fits-all care, research is concentrating on molecular and metabolic phenotyping (adipokine profiles, omics panels, and insulin sensitivity assays) to group individuals into pathways that are most likely to benefit from particular treatments.

Digital health, behavioral interventions, and integrated care

Access to multidisciplinary care (psychology, gynecology, endocrinology, and dietetics) can be enhanced by telemedicine, smartphone apps for tracking weight and cycles, and organized behavioral programs. It is becoming more and more advised to use integrated models that incorporate medication, lifestyle, and mental health assistance.

Reproductive technologies and fertility preservation

Reproductive possibilities are increased by advancements in ovulation induction, in vitro fertilization procedures, and fertility preservation counseling (for those postponing pregnancy). Research is still being done on adjunctive pharmacologic techniques to lower OHSS and enhance oocyte quality.

Novel pharmacologic agents & repurposing

Inositols, myo- and D-chiro-inositol formulations, insulin sensitizers other than metformin, and selective androgen receptor modulators for targeted hyperandrogenism are some of the other alternatives being studied. There is still a need for large, carefully planned randomized trials.

Challenges and gaps in care

1. **Diagnostic heterogeneity:** There have been recurrent efforts to standardize definitions for clinical and scientific purposes since different diagnostic criteria have an impact on prevalence estimates and clinical paths.
2. **Limited longitudinal data:** Better data is needed to determine the long-term cardiometabolic and reproductive trajectories under various treatments, even if the short-term results of numerous interventions are known.
3. **Access & equity:** Inequalities in access to specialized fertility services, multidisciplinary treatment, and costly drugs (such GLP-1 RAs) restrict equitable outcomes.
4. **Mental-health integration:** Despite the high frequency of mood disorders, psychological care frequently lacks enough funding.
5. **Pregnancy and safety data:** The safety of newer agents (such as GLP-1 RAs and weight-loss medications) during conception and pregnancy is not fully established, necessitating careful counseling.
6. **Implementation gaps:** Due to fragmented care, physician knowledge gaps, and budget limitations, guidelines frequently do not become standard practice.

DISCUSSION

One well-known instance of a chronic, multisystemic condition with clinical implications that go well beyond gynecology is polycystic ovarian disease (PCOD). The results compiled in this study highlight three characteristics that define the opportunity and the difficulty in PCOS care: substantial psychosocial load, interwoven reproductive–metabolic pathophysiology, and clinical heterogeneity. Together, these characteristics make diagnosis, treatment choices, long-term risk management, and the delivery of the health system more difficult, but they also make it obvious where advancements will have the biggest positive effects. PCOS can manifest in a variety of ways, including mixed phenotypes, largely metabolic (insulin resistance, dyslipidemia, obesity), or primarily reproductive (infertility, anovulation). A large portion of the variation in research findings and clinical reactions to treatment can be explained by this heterogeneity. The lesson for doctors is that phenotype-driven care is necessary: patients with hirsutism or menstrual control as their main concerns have different first-line priorities than those who present with metabolic syndrome and significant obesity. Heterogeneity necessitates stratified analysis and strong phenotyping for researchers; combining all PCOS patients without considering metabolic or reproductive subgroups would continue to obfuscate signals for targeted therapy and dilute detectable benefits.

CONCLUSION

PCOD presents a significant opportunity for bettering women's health as well as a clinical issue. Phenotype-guided, integrative approaches that concurrently address reproductive goals, metabolic risk, and psychological wellbeing are the way of the future for PCOD therapy. Though their promise will only be fulfilled by thorough long-term research, safety monitoring in reproductive contexts, and intentional efforts to make effective care accessible and egalitarian, emerging treatments and digital care models have the potential to significantly improve outcomes. Clinicians should continue to screen for and manage metabolic risk, focus lifestyle changes, offer evidence-based reproductive therapies (such as letrozole for ovulation induction when warranted), and incorporate mental health support into standard care.Though many patients might benefit from more accessible and efficient metabolic treatments and integrated models of care, established therapy (lifestyle, COCs, metformin, and letrozole for ovulation induction) are still necessary. Though more thorough long-term data and equitable implementation are required, emerging strategies—most notably GLP-1 receptor agonists for weight and metabolic control, precision phenotyping, and digital integrated care—offer promising paths to lessen burden. For women with PCOD to benefit from these advancements, coordinated clinical, scientific, and policy initiatives are needed.

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