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Review Article

PCOD vs PCOS: A Comparative Review

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ABSTRACT

Polycystic Ovary Syndrome (PCOS) and Polycystic Ovarian Disease (PCOD) are common hormonal disorders affecting women of reproductive age. Though they share similar symptoms like irregular periods, weight gain, and acne, they differ in causes, severity, and treatment. PCOS is more serious, linked to high androgen levels, infertility, and long-term health risks like diabetes and heart disease. PCOD is milder and often reversible with lifestyle changes. This review compares PCOS and PCOD in terms of symptoms, causes, diagnosis, and treatment options, including modern medicine and Ayurvedic remedies. It also highlights the importance of diet, exercise, and sleep in managing these conditions.

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) and Polycystic Ovarian Disease (PCOD) are common endocrine disorders among women of reproductive age, with an estimated prevalence of 5 -10%1. “PCOD, also referred to as Stein-Leventhal Syndrome2, it is characterized by the development of multiple small, fluid-filled cysts within the ovaries”. These cysts typically contain immature or partially developed eggs. Both genetic predisposition and environmental factors play a role in the onset of this disorder. Symptoms often include irregular menstrual cycles, weight gain, acne, and changes in physical appearance. If left untreated, PCOD can lead to serious health concerns such as type 2 diabetes, cardiovascular disease, obesity, mood disturbances, endometrial cancer, and sleep apnea.3 PCOS, on the other hand, is considered the most prevalent endocrine disorder among reproductive-aged women. It involves enlarged ovaries with numerous cysts along the outer edge and is often associated with metabolic disturbances. Compared to PCOD, PCOS is more severe due to the presence of excess androgen (male hormone) production, which leads to hormonal imbalance, anovulation, and an

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increased risk of infertility and ovarian complications4. In the current Indian scenario, the incidence of both conditions is steadily increasing. Although both PCOD and PCOS involve cyst formation in the ovaries, the presence of cysts is not always a universal feature, particularly in PCOS. In PCOD, the ovaries typically release immature or partially mature eggs, while PCOS results in the overproduction of androgens, heightening the risk of excessive cyst formation and even ovarian cancer.5 A regional study conducted in Southern India and Maharashtra reported that PCOD affects approximately 22.5% of menstruating women, while PCOS has a prevalence rate of 9.13%. Fertility outcomes also vary between the two: Women with PCOD can often conceive with minimal medical assistance and lifestyle adjustments. However, conception in PCOS is more challenging due to pronounced hormonal disruption6.

Difference Between PCOD and PCOS

Characteristic	PCOS	PCOD
Occurrence	Rare And Lesser Patients	Common In Women Worldwide
Definition	Endocrine Disorder of Ovaries with Immature Follicles	Metabolic Disorder Due To Hormonal Imbalance
Presence Of Cysts	Minimum Number of Cysts	More Than 10 Cysts in Each
Cause	Genetic, Lifestyle, Eating Habits, Mental Health	Genetic, Insulin Resistance, Inflammation, Weight
Severity	PCOS Cannot Be Revered	It Can Be Revered with Medication
Treatment	Diet Control, Exercise, Lifestyle Change and Medication	Diet, Exercise, Medication, Surgery
Complication	Difficultyin Conception, Risk of Diabetes, High Blood Pressure, Obesity, Endometrial Cancer	Conception Achievable with Medical Aid
Androgen Level	Elevated Androgen Level, Leading to Symptom Like Hirsutism, Acne, And Scalp Hair Thinning	Androgen Levels May Be Slightly Elevated, But Not ToThe Extent Seen In PCOS
Symptoms	Weight Gain, Hirsutism, Acne, Metabolic Disturbance	Irregular Periods, Mild Acne and Weight Gain

Epidemiology

Polycystic Ovarian Disease (PCOD) affects approximately 5–10% of women of reproductive age and is considered a common condition. However, it is often underdiagnosed compared to PCOS due to milder or less noticeable symptoms. Women with PCOD may experience incomplete anovulation, irregular menstrual cycles, or multiple ovarian cysts. Symptoms typically begin during adolescence and may persist into the third decade of life, prompting many to seek medical attention for menstrual irregularities. Lifestyle factors such as poor diet, high stress, and physical inactivity especially prevalent in urban settings are associated with PCOD. While many women with PCOD are at higher risk of obesity, they tend to have fewer metabolic complications than those with PCOS. Underdiagnosis is more common in regions with limited healthcare access or low awareness[7,8]. Polycystic Ovary Syndrome (PCOS) is significantly more prevalent and extensively studied compared to Polycystic Ovary Disease (PCOD), with a global incidence ranging from 6% to 20%, depending on diagnostic criteria and population differences. PCOS encompasses a spectrum of clinical features such as chronic anovulation, hyperandrogenism, insulin

resistance, and obesity. These symptoms often begin during adolescence, marked by irregular menstruation, acne, or hirsutism, and may progress to affect fertility and long-term health. Genetic predisposition plays a major role, with higher prevalence and more severe metabolic consequences observed among South Asian women, including increased risk of type 2 diabetes. Lifestyle factors like sedentary behavior and unhealthy diets further exacerbate the condition, particularly in urbanized and industrialized settings. Due to its broad impact on reproductive, metabolic, and cardiovascular health, PCOS is now recognized as a more serious and clinically significant disorder than PCOD[9,10].

Pathophysiology

High Levels of Insulin in PCOS: Insulin, a polypeptide hormone produced by pancreatic beta cells, primarily functions to reduce blood glucose levels. Elevated insulin levels in PCOS indicate its role in both metabolic and reproductive dysfunctions. In cases where insulin is not produced adequately, blood sugar remains high, contributing to hyperandrogenism. Additionally, insulin resistance may occur, where insulin is produced but not effectively utilized by the body. This elevated insulin stimulates the ovaries to produce more androgens, such as testosterone, which disrupts ovulation. In PCOS-related pregnancies, excess testosterone is not regulated effectively. Hyperinsulinemia may also lead to increased androgen levels and reduced sex hormone-binding globulin, further worsening the hormonal imbalance. Moreover, peripheral insulin resistance is linked to uterine and ovarian complications[11].

Ovarian fibrosis: In PCOS, ovarian fibrosis is marked by thickening due to excessive collagen deposition. Transforming Growth Factor-beta1 (TGF-β1) is a key profibrotic mediator elevated in PCOS ovaries, promoting extracellular matrix (ECM) remodeling. Connective Tissue Growth Factor (CTGF), along with TGF-β1, plays a pivotal role in granulosa cell (GC) signaling. Endoplasmic Reticulum (ER) stress activates TGF-β1 and CTGF expression, contributing to interstitial fibrosis. Experimental models confirm that HA-induced ER stress triggers TGF-β1 signaling and fibrotic changes. Agents like TUDCA and adrenomedullin can attenuate ovarian fibrosis by reducing ER stress and profibrotic markers. Elevated levels of TGF-β1 and CTGF are consistently observed in PCOS models. These findings suggest ER stress–TGF-β1 axis as a pathogenic mechanism in ovarian fibrosis. The persistent fibrotic environment affects follicular function and contributes to PCOS-associated infertility. Overall, ER stress–mediated TGF-β1 signaling is central to the etiology of ovarian interstitial fibrosis in PCOS[12].

Genetics: Genetic predisposition is a significant risk factor for PCOS. Variations such as single nucleotide polymorphisms (SNPs), including alterations, deletions, and inversions, contribute to its development. For example, SNP rs2414096 in the CYP19 gene has been linked to PCOS, while SNP rs4077582 in the CYP11A1 gene is associated with increased androgen levels, worsening the condition. Additionally, polymorphisms in the CYP19A1 gene have been related to a higher risk of endometrial and breast cancers[13].

Obesity: Obesity plays a pivotal role in the development and progression of Polycystic Ovary Syndrome (PCOS). It is often driven by poor lifestyle choices, such as high intake of calorie-dense and processed foods, leading to metabolic dysfunction. Increased adiposity contributes to insulin resistance and elevated circulating insulin levels, which in turn stimulate excessive androgen

production by the ovaries. This hormonal imbalance is a hallmark feature of PCOS, aggravating symptoms like anovulation, hirsutism, and menstrual irregularities. Furthermore, obesity is closely associated with dyslipidemia, further complicating the endocrine profile of affected women. It also increases the risk of comorbidities such as type 2 diabetes and cardiovascular diseases. Beyond metabolic effects, obesity raises the likelihood of hormone-dependent malignancies, including breast and endometrial cancers. Thus, managing obesity is essential in both prevention and therapeutic strategies for PCOS[14].

Fetal Androgen Exposure in PCOS: The developmental hypothesis of PCOS suggests that elevated androgen exposure during fetal life contributes to the syndrome's onset later in life. This exposure may disrupt normal development of steroidogenesis, insulin signaling, pancreatic β-cell function, and neuroendocrine regulation. Genetic predisposition may cause fetal and early-life ovaries to secrete excess androgens. Studies indicate that prenatal androgen exposure programs long-term metabolic, reproductive, and neuroendocrine abnormalities. Evidence also links daughters of women with PCOS to increased anogenital distances, a marker of prenatal androgen influence. However, how fetal androgen exposure occurs despite protective mechanisms like placental aromatase and maternal SHBG remains unclear[15].

Drug Used in PCOD and PCOS

When initial lifestyle interventions are inadequate or unsuccessful, pharmacological treatment should be considered. Upon appropriate clinical assessment, therapy should be initiated to effectively manage symptoms and reduce the risk of long-term complications[16].

Metformin: Metformin, commonly used for managing glucose intolerance in PCOS, does not significantly improve live birth rates or ovulation when used alone or with clomiphene citrate. It is not recommended solely for inducing ovulation. However, when combined with low-dose gonadotropins in timed intercourse, metformin has shown improved clinical pregnancy and live birth rates, along with reduced ovarian hyper-responsiveness and cycle cancellations. Despite these benefits, it does not affect rates of miscarriage, multiple pregnancies, or OHSS consistently. In assisted reproductive techniques (ART), metformin may help reduce OHSS risk and improve endometrial receptivity, but its overall impact on live birth outcomes remains uncertain. Routine use in IVF is not supported unless insulin resistance or glucose issues are present[17].

Glucocorticoids: Glucocorticoids like prednisone and dexamethasone are employed to trigger ovulation, especially in clomiphene citrate (CC)-resistant PCOS cases. Elnashar et al. reported that adding short-term, high-dose dexamethasone improves ovulation and pregnancy rates without negatively impacting the endometrium in women with normal DHEAS levels. For those with elevated adrenal androgens, low-dose dexamethasone (0.25–0.5 mg) at bedtime is effective. A study involving 230 CC-resistant women showed improved ovulation when 2 mg dexamethasone was administered from day 5 to 14 of the cycle. However, due to potential negative effects on insulin sensitivity, prolonged glucocorticoid use is not advised[18].

Orlistat: Orlistat, a gastric and pancreatic lipase inhibitor, works by blocking the breakdown and absorption of dietary fats, thereby reducing triglyceride hydrolysis. Originally approved for obesity treatment, it has been evaluated in multiple clinical trials involving women with PCOS.

Findings from three of these studies suggest that orlistat enhances insulin sensitivity, lowers insulin levels, and contributes to a significant reduction in BMI. These outcomes highlight its potential role in improving metabolic profiles in PCOS patients[19].

Clomiphene: Clomiphene citrate remains the primary pharmacological agent for initiating ovulation in PCOS patients, despite its unclear mechanism of action. Typically, treatment begins with 50 mg/day for 5 days; if ovulation does not occur, the dose can be increased to 100 mg/day after a 30-day interval. While further therapy is generally limited to three cycles, up to six cycles may be attempted. Clomiphene induces ovulation in many cases, yielding pregnancy in about 30%, though 20% may end in miscarriage or stillbirth. Side effects include ovarian enlargement, OHSS, multiple gestations, vasomotor symptoms, and gastrointestinal discomfort[20].

Oralcontraceptive: Oral contraceptives (OCs) are commonly recommended for women with PCOS who are not seeking pregnancy. Their primary mode of action involves regulating menstrual cycles and lowering androgen levels, which helps alleviate symptoms like acne and hirsutism. Combined estrogen–progestin formulations are the most frequently used OCs for managing these clinical manifestations. Newer OCs containing anti-androgenic progestins such as drospirenone (e.g., Yaz) and dienogest (e.g., Natazia) show enhanced efficacy in addressing hyperandrogenic symptoms. Clinical improvements, particularly in hirsutism, are typically observed after around six months of consistent treatment[21].

Ayurvedic Treatment

Cinnamon: Cinnamon (Cinnamomum zeylanicum), known as Kalmi-Dalchini, is a potent natural remedy for PCOS due to its ability to enhance insulin sensitivity. Its high fiber content helps regulate the menstrual cycle and curb appetite. Clinical trials show cinnamon significantly reduces insulin resistance without side effects. It also improves lipid profiles and offers antioxidant benefits. Overall, it supports hormonal balance and reduces PCOS-related risks[22].

Panax Ginseng: Panax ginseng, a traditional herb from the Araliaceae family, is rich in ginsenosides like Rb1, Rc, and Rd, known for anti-aging effects. It serves as a potent dietary supplement and hormonal modulator. In animal models of PCOS, ginseng improved ovarian morphology. Kampo formulations containing ginseng reduced plasma LH levels. This highlights its potential in managing ovulatory dysfunction in PCOS. *Summary for Review Article (5 lines)[23].

Ashwagandha: Ashwagandha (*Withania somnifera*) is a medicinal shrub known for its adaptogenic and anti-stress effects. It modulates cortisol levels, promoting emotional balance and reducing anxiety, stress, and mood swings.The herb's bioactive alkaloids, like withanine and somniferine, exhibit anti-inflammatory actions.It supports hormonal harmony by minimizing disruption in progesterone and thyroid levels. Ashwagandha's endocrine-regulating properties make it valuable in managing reproductive and menstrual health[24].

Fennel: Foeniculum vulgare (Shatapushpa) is recognized for its therapeutic role in PCOS management.Its seeds are abundant in phytoestrogens, which aid in lowering insulin resistance. The anti-inflammatory properties of fennel contribute to mitigating PCOS-related symptoms. It helps correct cellular imbalances that trigger metabolic irregularities in PCOS. Fennel acts as a natural endocrine

modulator,supporting hormonal and metabolic equilibrium. drug[25].

Liquorice: Glycyrrhiza glabra (liquorice), from the Leguminosae family, exhibits antifungal, antiviral, and antihyperglycemic effects. Its key bioactive agent, glycyrrhizic acid, and phytoestrogens like liquiritigenin and glabridin contribute to its therapeutic role. Liquorice influences vascular function and shows estrogen-like activity. Certain compounds aid weight loss and reduce androgen levels. Combined with spironolactone, it may enhance PCOS treatment by lowering androgenic symptoms[26].

Non - Pharmacological Treatment

Lifestyle Modification for Managing PCOD: Lifestyle modification remains the primary and most effective approach in managing PCOD, though it should complement rather than replace medical treatment. Clinical guidelines for various metabolic and hormonal conditions stress the importance of maintaining an active lifestyle, achieving a healthy body weight, adopting balanced dietary habits, and avoiding tobacco use. These behavioral changes not only aid in managing symptoms but also improve both physical and mental health over time. While such adjustments are not an immediate cure, they provide a sustainable foundation for long-term wellness. Nutritional counseling has also been widely recommended for women with PCOD. However, research suggests that isocaloric diets alone without combining with exercise may not yield significant biochemical or anthropometric improvements, and extreme calorie restriction has shown limited benefits as well[27].

Sleep: Sleep irregularities are frequently observed among individuals with PCOD, and studies indicate a strong association between these disturbances and mental health challenges such as anxiety and depression. Inadequate sleep duration has also been linked to a higher likelihood of developing insulin resistance, obesity, and type 2 diabetes. The mechanisms underlying this relationship may involve several physiological pathways, including disruptions in autonomic function and hormonal imbalances in appetite-regulating hormones like ghrelin and leptin. Additionally, melatonin concentrations tend to be reduced in women with PCOD, particularly within their follicular fluid. Melatonin receptors in the ovaries, along with its presence in intrafollicular fluid, play an essential role in regulating sex steroid secretion during follicular development. Furthermore, melatonin's antioxidant properties contribute to the protection of ovarian follicles against oxidative stress. The circadian rhythm of melatonin secretion typically peaking at night and declining during the day helps synchronize the body's light-dark communication system. This pattern contrasts with that of cortisol, which peaks in the morning and decreases during sleep, as it is controlled by the hypothalamic-pituitary-adrenal (HPA) axis[28].

Physical Activity: Engaging in regular physical activity, particularly aerobic exercises, significantly enhances insulin sensitivity, promotes glucose metabolism, and supports overall health. Recent evidence highlights that the intensity of exercise may have a greater influence on metabolic outcomes than the volume of activity performed. In the context of PCOS, consistent exercise contributes to improved reproductive health, reduced risk of weight gain and metabolic syndrome, better mental well-being, and an overall improvement in quality of life. Although specific recommendations for exercise type, intensity, and duration may differ between individuals, aiming for at least 120 minutes of aerobic exercise per week is generally considered an effective starting point[29].

Dietary Interventions: Caloric reduction and limiting total fat intake are cornerstone recommendations for managing weight and PCOS symptoms, although the superiority of any single diet type remains debated. Low-carbohydrate diets, when consumed for less than 45% of total daily calories, have shown promise in improving serum lipid profiles and total cholesterol levels. Furthermore, dietary adjustments promoting PCOS symptom relief, such as those that increase levels of follicle-stimulating hormone (FSH) and sex-hormone-binding globulin SHBG, are also being studied, though the precise mechanism remains to be fully elucidated. The text notes that metformin, an established pharmacological agent, combined with lifestyle modifications, has shown superior results compared to either intervention alone, particularly in improving insulin resistance and reducing circulating androgen levels. Ultimately, dietary strategies effective for managing PCOS are those that are sustainable for long-term adherence and contribute to maintaining a healthy body mass index (BMI)[30].

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