CODEN [USA]: IAJPBB ISSN : 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES

SJIF Impact Factor: 7.187

https://doi.org/10.5281/zenodo.17141498

https://www.iajps.com/volumes/volume12-september-2025/14-issue-09-september-25/

Available online at: http://www.iajps.com *Review Article*

POLYCYSTIC OVARIAN SYNDROME: A SHORT REVIEW

Anupa M Chandran [1], Jayachandran T P [2], Varsha V [3]

[1], [3] Student, M Pharm, Department of Pharmaceutical Sciences, CPAS, Cheruvandoor campus, Ettumanoor P.O, Kottayam,686631, Kerala, India

[2] Associate Professor, Department of Pharmaceutical Sciences, CPAS, Cheruvandoor campus, Ettumanoor P.O, Kottayam,686631, Kerala, India

Abstract:

Polycystic Ovarian Syndrome (PCOS) is a highly prevalent and complex endocrine disorder affecting reproductive-aged women globally, characterized by a triad of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology. This review provides a comprehensive overview of the pathogenesis, clinical manifestations, diagnostic criteria, and management strategies for PCOS. The etiology is multifaceted, involving a complex interplay of genetic, metabolic, and environmental factors, with insulin resistance and hyperandrogenism being the central pathophysiological drivers. Clinically, PCOS presents with a wide spectrum of symptoms, ranging from reproductive issues like menstrual irregularities and infertility to androgenic signs such as hirsutism and acne. The diagnosis primarily relies on the Rotterdam criteria, which require the presence of at least two of the three cardinal features. Beyond its reproductive and aesthetic impact, PCOS is a significant risk factor for a host of serious long-term comorbidities, including metabolic syndrome, type 2 diabetes, cardiovascular disease, and endometrial cancer. The management approach is highly individualized and aims to address the patient's primary concerns while mitigating future health risks. Treatment strategies encompass a multi-pronged approach, including lifestyle modifications such as diet and exercise as first-line therapy, followed by pharmacological interventions like oral contraceptives, anti-androgens, and insulin-sensitizing agents such as metformin. This review underscores the critical need for early diagnosis and a holistic, patient-centered approach to care to improve the quality of life and long-term health outcomes for women with PCOS.

Keywords: *Polycystic Ovarian Syndrome, Hyperandrogenism, Insulin Resistance, Infertility, Menstrual cycle*

Corresponding author:

Anupa M Chandran,

Student, M Pharm, Department of Pharmaceutical Sciences, CPAS, Cheruvandoor campus, Ettumanoor P.O, Kottayam,686631, Kerala, India

QR CODE

*Please cite this article in **press** Anupa M Chandran et al., **Polycystic Ovarian Syndrome: A Short Review**, Indo Am. J. P. Sci, 2025; 12(09).*

INTRODUCTION:

Polycystic ovarian syndrome (PCOS) is a heterogeneous endocrine disorder distinguished by the manifestation of ovarian cysts, anovulation, and endocrine variation that severely impacts the life of a woman. The regular menstrual cycle is disrupted by the disruption of reproductive hormones such as LH, FSH, estrogen, and testosterone, which can result in abnormalities, amenorrhea, and oligomenorrhea. The World Health Organization (WHO) estimates that PCOS affects about 116 million women globally, or 3.4% of the total population. Although there are significant individual variances, PCOS is diagnosed with hyperandrogenism, irregular menstruation, and varied diameters of ovarian cysts (1).

There is still debate in clinical endocrinology regarding the diagnosis of PCOS. The National Institutes of Health (NIH) criteria were established in 1990 to provide a comprehensive and detailed description for the diagnosis of PCOS. Then, in 2003, a Rotterdam workshop created a brand-new diagnostic standard called the Rotterdam criteria. The presence of two of the three conditions—oligomenorrhea/anovulation, clinical/biochemical hyperandrogenism, and polycystic ovaries (≥12 follicles in each ovary measuring 2–9 mm)—is required to meet this criteria. The diagnostic standards were updated by the Androgen Excess Society (AES) in 2006. Clinical/biochemical hyperandrogenism along with either polycystic or oligoanovulation ovaries is a prerequisite for the AES (2). PCOS-afflicted women are more likely to experience emotional problems, obstetrical issues, endometrial cancer, infertility, and metabolic abnormalities, including type 2 diabetes. These women are also likely to be at higher risk for ovarian cancer, venous thromboembolism, and cardiovascular and cerebrovascular events (3). Infertility, mood swings, hirsutism, obesity, acne, and pattern hair loss are among the issues that women with PCOS may face, in addition to aberrant insulin activity. A dysregulated hormonal state, hyperandrogenism, hyperinsulinemia, and inflammation are the results of PCOS, a condition that is frequently brought on by a poor lifestyle, neuroendocrine disorders, hereditary factors, and androgen exposures (4).

This review aims to provide a comprehensive, evidence-based synthesis of the current understanding of Polycystic Ovarian Syndrome (PCOS). We will first discuss the complex pathophysiology and diverse clinical manifestations, followed by an in-depth examination of the diagnostic criteria and associated comorbidities. Finally, we will outline the latest evidence-based management strategies, from lifestyle modifications to pharmacological treatments, to provide a valuable resource for clinicians and researchers. This work highlights the critical need for a holistic approach to care to improve the long-term health outcomes and quality of life for women with PCOS.

EPIDEMIOLOGY

Polycystic Ovarian Syndrome is a condition that many women in their reproductive years have. The age range of the population, the sort of diagnostic criteria used, and ethnicity all affect the prevalence of PCOS. More research indicates that this issue does not just impact young women; it can also start in adolescence (5). The percentage of teenage girls between the ages of 15 and 19 who had been diagnosed (and proven to have) PCOS-NIH was 0.56% (95% CI, 0.52%–0.60%). Including all cases with a PCOS diagnosis, independent of manual confirmation by chart review, resulted in a 0.78% (95% CI, 0.72%–0.81%) prevalence of PCOS (6). The diagnostic criteria, phenotypes, and demographics under study all affect PCOS prevalence. The prevalence rates of PCOS were 6–10% and 10%, respectively, based on the Rotterdam and National Institutes of Health diagnostic criteria.1 The Rotterdam criteria showed a prevalence range of 8–13% (7). A higher prevalence of PCOS is linked to a number of conditions, such as obesity34, insulin resistance35, type I or type II diabetes mellitus36–38, or oligo-ovulatory infertility.3,39 Additionally, it appears that Mexican American women are more likely to have PCOS than White or African American women (8).

PATHOPHYSIOLOGY

The underlying pathophysiology of PCOS is still not fully understood. There are probably several underlying pathophysiologic pathways due to the disorder's variability. The pathophysiology of PCOS has been explained by several theories: 1) Hyperinsulinemia and insulin resistance are caused by changes in insulin production and action. 2) Increased luteinizing hormone (LH) secretion is the outcome of a change in gonadotropin-releasing hormone secretion. 3) An increase in ovarian androgen production due to a malfunction in androgen synthesis (9).

The pathophysiology of PCOS's hyperandrogenism appears to be significantly influenced by compensatory hyperinsulinemia, which is a result of insulin resistance in the condition. Acanthosis nigricans is caused by excessive skin basal cell development, aberrant hepatic and peripheral lipid metabolism, and the stimulation of androgen release by the ovarian theca by hyperinsulinemia (10).

PCOS-afflicted women produce more luteinizing hormone (LH) due to relative follicle-stimulating hormone (FSH) deficiency, sustained high LH (and consequently gonadotropin-releasing hormone

[GnRH]) pulse frequency, increased LH pulse amplitude, and exaggerated LH responses to exogenous GnRH. These gonadotropin secretion anomalies significantly contribute to PCOS's ovarian hyperandrogenism and ovulatory dysfunction. Long-acting GnRH agonists significantly lower androgen production in PCOS-afflicted women, and ovarian hyperandrogenemia in PCOS is LH-dependent. PCOS usually appears at or soon after the pubertal rise in LH secretion. More recently, genes associated with gonadotropin, such as those for FSH subunit beta (FSHB), the FSH receptor (FSHR), LH subunit beta (LHB), and the LH/choriogonadotropin receptor (LGCGR), have been linked to the development of PCOS(11).

One of the crucial traits of PCOS patients is the irregularity of their menstrual cycle. As the menstrual cycle lengthens, anovulation increases in frequency, contributing to amenorrhea, endometrial hyperproliferation, and even the development of cancer. The duration of the menstrual cycle and the blood level of testosterone have a positive correlation, indicating that hyperandrogenism may contribute to the onset and advancement of PCOS. In female patients, hyperandrogenism may also be a risk factor in and of itself for metabolic syndrome, obesity, cardiovascular disease (CVD), and type 2 diabetes (12).

DIAGNOSIS

The Rotterdam PCOS Diagnostic Criteria for adult women were recommended by the recommendations. Crucially, PCOS is an excluding diagnosis. It is necessary to rule out other conditions with comparable clinical characteristics when making a diagnosis. According to the Rotterdam criteria, adult women had to meet two of three requirements: (1) oligo-anovulation; (2) hyperandrogenism, either biochemical or clinical; and (3) ultrasound-detected polycystic ovary shape. An ultrasound is not required for diagnosis when an adult woman has both hyperandrogenism and ovulatory failure. To diagnose PCOS in a teenage female, there must be persistent clinical and/or biochemical hyperandrogenism in addition to ovulatory failure, which is now precisely characterized by time after menarche. The guidelines are significant because they emphasize that ultrasound investigations to evaluate for polycystic morphology are not required until 8 years after menarche because of the lack of sensitivity and specificity at this stage of life (15). Severe acne and hirsutism are symptoms of clinical hyperandrogenism. To validate biochemical hyperandrogenism, high-quality testosterone tests are necessary (13).

MANAGEMENT AND TREATMENT

Lifestyle modifications

According to a recent study, a whole lifestyle shift, including nutritional adjustments, may be more successful than diet alone at controlling PCOS symptoms. Diet alone would be useless unless a lifestyle adjustment approach is incorporated, since women with PCOS often have more body fat than average women, with most of that fat located beneath the hip and abdomen. The best veggies for PCOS patients are green leaves, but other dietary possibilities include legumes, pulses, beans, and fruits, including apples, guavas, and grapes (14).

Pharmacological interventions

- **Oral contraceptives (OCPs)**

The OCPs are separated into progesterone-only tablets and mixed pills that include progesterone (norethisterone, desogestrel) and estrogen (estradiol dosage up to 50µg). They are the first line of treatment for women who experience irregular menstruation and do not wish to ovulate. OCP usage reduces the incidence of ovarian malignancies, while women with PCOS are more likely to develop tumors (1).

- **Metformin**

Metformin is a biguanide medication used orally to treat diabetes. Metformin is recommended as the first-line therapy for cutaneous symptoms and pregnancy problems in women with PCOS, despite conflicting research on its effects. In individuals who are resistant to clomiphene citrate, it is also used in conjunction with it to enhance reproductive results (15).

- **Inositol**

Due to their lack of adverse effects, new medications that have recently been offered as a novel treatment for PCOS are becoming increasingly well-known. These are two stereoisomers of the insulin-sensitizing chemical inositol: myo-inositol (MYO) and D-chiro-inositol (DCI). An increasing body of research indicates that insulin resistance may be brought on by a change in the metabolism of inositol phosphoglycans (IPG) mediators and second messengers, or by a malfunction in their tissue availability (16).

- **Clomiphene citrate**

A selective modulator of the estrogen receptor, clomiphene citrate (CC), is the most often used medication to stimulate ovulation in females with polycystic ovarian syndrome. In clinical practice, it increases the release of gonadotropin-releasing hormone by competitively antagonistically binding to the hypothalamic estrogen receptor. Increased pituitary synthesis and release of FSH, which promotes follicle development and recruitment, is the outcome of greater GnRH. Initially, 50 mg of CC should be taken orally every day for five days, starting on the second to the fifth day of the menstrual cycle. Five to ten days following the conclusion of therapy, ovulation often occurs. In

cases when ovulation did not occur in the previous cycle, the dosage might be raised by 50 mg per day (17).

- **Anti-Androgen Therapy**

A dose of 50–100 mg twice a day of spironolactone is an effective treatment for hirsutism. Because oral contraceptives and spironolactone have additive effects of androgen suppression and androgen blocking, they are frequently used in conjunction. During pregnancy, spironolactone should not be used due to the possibility of teratogenicity (18).

- **Thiazolodinediones**

Troglitazone improved glycemic indices, ovulation, hirsutism, and free testosterone levels in women with polycystic ovarian syndrome in a large randomized controlled study (18).

- **Vitamin D Supplementation**

Insulin resistance and diabetes, which result from vitamin D deficiency, cause hyperandrogenism and irregular menstruation. The importance of vitamin D in reproduction has also been shown. In the treatment of PCOS, supplements may be beneficial (19).

CONCLUSION:

In conclusion, Polycystic Ovarian Syndrome (PCOS) is a prevalent and multifaceted endocrine disorder. Its pathophysiology is driven by an intricate relationship between hyperandrogenism and insulin resistance, which leads to a wide range of clinical manifestations. Effective management requires a personalized, comprehensive approach that begins with lifestyle modifications and may include pharmacological interventions to address both symptoms and long-term health risks. Continued research is vital to further our understanding of PCOS and to develop more targeted therapies, ultimately improving the health and quality of life for women with this condition.

REFERENCES:

1. Bulsara J, Patel P, Soni A, Acharya S. A review: brief insight into polycystic ovarian syndrome. Endocrine and metabolic science. 2021 Jun 30;3:100085. https://doi.org/10.1016/j.endmts.2021.100085
2. Deswal R, Narwal V, Dang A, Pundir CS. The prevalence of polycystic ovary syndrome: a brief systematic review. Journal of human reproductive sciences. 2020 Oct 1;13(4):261-71. 10.4103/jhrs.JHRS_95_18.
3. Azziz R, Carmina E, Chen Z, Dunaif A, Laven JS, Legro RS, Lizneva D, Natterson-Horowtiz B, Teede HJ, Yildiz BO. Polycystic ovary syndrome. Nature reviews Disease primers. 2016 Aug 11;2(1):1-8. https://doi.org/10.1038/nrdp.2016.57.
4. Chaudhuri A. Polycystic ovary syndrome: Causes, symptoms, pathophysiology, and remedies. Obesity Medicine. 2023 May 1;39:100480. https://doi.org/10.1016/j.obmed.2023.100480
5. Jakubowska-Kowal KM, Skrzynska KJ, Gawlik-Starzyk AM. Prevalence and diagnosis of polycystic ovary syndrome (PCOS) in adolescents—what's new in 2023? Systematic review. Ginekologia Polska. 2024;95(8):643-9. DOI: 10.5603/gpl.98849
6. Christensen SB, Black MH, Smith N, Martinez MM, Jacobsen SJ, Porter AH, Koebnick C. Prevalence of polycystic ovary syndrome in adolescents. Fertility and sterility. 2013 Aug 1;100(2):470-7. https://doi.org/10.1016/j.fertnstert.2013.04.001
7. Neven AC, Laven J, Teede HJ, Boyle JA. A summary on polycystic ovary syndrome: diagnostic criteria, prevalence, clinical manifestations, and management according to the latest international guidelines. InSeminars in reproductive medicine 2018 Jan (Vol. 36, No. 01, pp. 005-012). Thieme Medical Publishers. 10.1055/s-0038-1668085.
8. Goodarzi MO, Azziz R. Diagnosis, epidemiology, and genetics of the polycystic ovary syndrome. Best practice & research Clinical endocrinology & metabolism. 2006 Jun 1;20(2):193-205. https://doi.org/10.1016/j.beem.2006.02.005
9. King J. Polycystic ovary syndrome. Journal of Midwifery & Women's Health. 2006 Nov 1;51(6):415-22. https://doi.org/10.1016/j.jmwh.2006.01.008
10. Azziz R. Polycystic ovary syndrome, insulin resistance, and molecular defects of insulin signaling. The Journal of Clinical Endocrinology & Metabolism. 2002 Sep 1;87(9):4085-7. https://doi.org/10.1210/jc.2002-021131
11. McCartney CR, Campbell RE. Abnormal GnRH pulsatility in polycystic ovary syndrome: recent insights. Current opinion in endocrine and metabolic research. 2020 Jun 1;12:78-84. https://doi.org/10.1016/j.coemr.2020.04.005
12. Ye W, Xie T, Song Y, Zhou L. The role of androgen and its related signals in PCOS. Journal of cellular and molecular medicine. 2021 Feb;25(4):1825-37. https://doi.org/10.1111/jcmm.16205
13. Witchel SF, Teede HJ, Peña AS. Curtailing pcos. Pediatric research. 2020 Jan;87(2):353-61. https://doi.org/10.1038/s41390-019-0615-1.
14. Shahid R, Mahnoor, Awan KA, Iqbal MJ, Munir H, Saeed I. Diet and lifestyle modifications for effective management of polycystic ovarian syndrome (PCOS). Journal of food biochemistry. 2022 Jul;46(7):e14117. https://doi.org/10.1111/jfbc.14117

15. Shermin S, Noor A, Jahan S. Polycystic ovary syndrome: a brief review with recent updates. Delta Medical College Journal. 2019;7(2):84-99. https://doi.org/10.3329/dmcj.v7i2.45567
16. El Hayek S, Bitar L, Hamdar LH, Mirza FG, Daoud G. Poly cystic ovarian syndrome: an updated overview. Frontiers in physiology. 2016 Apr 5;7:124. https://doi.org/10.3389/fphys.2016.00124
17. Rababa'h AM, Matani BR, Yehya A. An update of polycystic ovary syndrome: causes and therapeutics options. Heliyon. 2022 Oct 1;8(10).
18. Setji TL, Brown AJ. Polycystic ovary syndrome: diagnosis and treatment. The American journal of medicine. 2007 Feb 1;120(2):128-32. https://doi.org/10.1016/j.amjmed.2006.06.029
19. dehghani Firouzabadi R, Aflatoonian A, Modarresi S, Sekhavat L, MohammadTaheri S. Therapeutic effects of calcium & vitamin D supplementation in women with PCOS. Complementary therapies in clinical practice. 2012 May 1;18(2):85-8. https://doi.org/10.1016/j.ctcp.2012.01.005