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Plant-based approaches in the management of polycystic ovary syndrome: A review

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Abstract
Polycystic Ovary Syndrome (PCOS) is a complex metabolic and hormonal condition affecting many women of reproductive age. Conventional treatments often target symptoms individually and may carry side effects with long-term use. Recently, plant-based approaches have emerged as complementary strategies in PCOS management due to their natural bioactive compounds and minimal side effects. This review examines current evidence on the role of plant-based diets, herbal remedies, and phytochemicals in addressing key features of PCOS, including insulin resistance, hormonal imbalance, inflammation, and oxidative stress. Findings suggest that certain plant-based foods and herbs may help regulate menstrual cycles, improve metabolic parameters, and reduce androgen levels. Overall, plant-based interventions offer a promising, holistic approach to supporting women with PCOS.

Keywords: PCOS, hormonal imbalance, insulin resistance, menstrual irregularities, ovarian dysfunction, infertility, metabolic disturbances, herbal treatment, plant-based therapy.

Introduction
Polycystic Ovary Syndrome (PCOS) is recognized as one of the most prevalent endocrine disorders among women of reproductive age, typically between 18 and 44 years. It is a leading cause of subfertility, particularly due to ovulatory dysfunction, which is present in approximately 70% of affected women. The syndrome disrupts normal ovarian function, often due to the presence of multiple small cysts, resulting in hormonal imbalances and irregular or absent ovulation.

PCOS is a chronic and multifaceted condition that manifests through a wide range of symptoms. These may include weight gain, high blood pressure, insulin resistance or type 2 diabetes, oily skin or dandruff, dark patches of skin (acanthosis nigricans, often on the neck and underarms), chronic pelvic pain, and acne. Elevated androgen levels are a hallmark of the disorder, leading to hirsutism, scalp hair thinning, male-pattern baldness, and acne. Reproductive symptoms involve irregular menstrual cycles, infrequent or absent ovulation, irregular uterine bleeding, and the presence of immature ovarian follicles.

Clinically, PCOS may also be referred to as hyper-androgenic anovulation (HA) or Stein-Leventhal syndrome. Diagnostic criteria often include the detection of ovarian enlargement (≥10 mL) and multiple small follicles (2-9 mm in diameter) in one or both ovaries. The condition is frequently diagnosed only after notable symptoms such as hair loss, infertility, or metabolic disturbances impact a woman's quality of life.

Epidemiological data show that, according to NIH diagnostic guidelines, between 4-10% of women of reproductive age globally may have PCOS. The World Health Organization (WHO) reported that in 2012, the global prevalence was over 116 million women, representing approximately 3.4% of the female population. In India, prevalence is particularly high, with estimates suggesting that 1 in 5 women are affected. A nationwide survey conducted in 2020 indicated that about 16% of Indian women aged 20 to 29 were diagnosed with PCOS.

Globally, around 1.55 million cases of PCOS were recorded in 2017 among women aged 15-49 years, with a 4.47% increase in incidence from 2007 to 2017. The age-standardized incidence rate reached 82.44 per 100,000, contributing to approximately 0.43 million disability-adjusted life years (DALYs). While symptoms often appear soon after menarche, most diagnoses occur between the ages of 20 and 30.

Emerging research suggests that PCOS may originate during fetal development, challenging the earlier notion that it is solely an adult-onset disorder.

As a lifelong condition, PCOS requires timely diagnosis and long-term management to mitigate its reproductive, metabolic, and psychological impacts [1, 2, 3].

Etiology and Pathophysiology

Hyperandrogenism and Follicular Arrest in PCOS

Hyperandrogenism (HA) is a central feature of polycystic Ovary Syndrome (PCOS) and contributes to hallmark symptoms such as hirsutism, acne, and androgenic alopecia. Elevated androgen levels, particularly free testosterone, are commonly observed in PCOS and are directly correlated with the condition's severity. The excess androgen production results primarily from abnormalities in ovarian and adrenal steroidogenesis, which are influenced by neuroendocrine and metabolic dysregulation.

Pathophysiological Consequences of Hyperandrogenism:

1. **Reduced SHBG Levels:** High androgen levels suppress sex hormone-binding globulin (SHBG), leading to increased circulating free testosterone.
2. **Peripheral Conversion in Adipose Tissue:** In fat tissue, testosterone is converted to estrone, which is further metabolized to estradiol, disrupting the estrogen balance.
3. **Impaired Follicular Development:** An abnormal estrone-to-estradiol ratio alters the LH: FSH ratio, resulting in ovulatory dysfunction.
4. **Increased Anti-Mullerian Hormone (AMH):** Elevated AMH inhibits the growth and maturation of ovarian follicles.
5. **Low Insulin-Like Growth Factor II (IGF-II):** Reduced IGF-II levels in follicular fluid impair estradiol synthesis and follicular progression.

These disruptions collectively lead to follicular arrest, a key feature in anovulatory PCOS.

Clinical manifestations of hyperandrogenism

Hirsutism

Hirsutism, defined by the growth of coarse, terminal hair in areas typical of male patterns (face, chest, back), is the most prominent sign of hyperandrogenism and affects about 60% of women with PCOS. Its severity varies significantly depending on ethnicity, necessitating population-specific diagnostic criteria. The modified Ferriman-Gallwey (mFG) scoring system is the standard tool used to assess hirsutism. It grades hair growth on nine body sites from 0 (no hair) to 4 (extensive growth). A total score of ≥ 7 is diagnostic, with scores of 7-9 indicating mild, 10-14 moderate, and ≥ 15 severe hirsutism.

Pathophysiologically, hirsutism results from increased free testosterone and heightened activity of 5-α reductase, which converts testosterone into dihydrotestosterone (DHT) a more potent androgen that directly stimulates hair follicles.

Acne

Acne is present in approximately 12-14% of PCOS patients and is more prevalent in certain populations, particularly Indo-Asians, while being less frequent in Pacific Islanders. The condition is driven by excess sebum production and clogged hair follicles, often leading to comedones, which may become inflamed due to the activity of cutibacterium acnes. This can progress into inflammatory acne lesions such as papules, pustules, or nodules.

Although androgens are known to increase sebum production, not all women with acne have biochemical hyperandrogenism. This suggests variability in androgen receptor sensitivity in the skin. Likewise, the presence of hirsutism does not necessarily imply acne, underscoring tissue-specific differences in androgen response.

Androgenic Alopecia

Hair thinning, or androgenic alopecia, in PCOS is characterized by diffuse hair loss over the vertex of the scalp while typically sparing the frontal hairline. The extent of hair loss differs among individuals and is frequently associated with seborrhea and scalp flaking [4].

Neuroendocrine and molecular mechanisms of hyperandrogenism in PCOS

Hyperandrogenism (HA) plays a pivotal role in disrupting the neuroendocrine regulation of the reproductive axis in polycystic ovary syndrome (PCOS). Elevated androgen levels interfere with the negative feedback mechanisms of estradiol and progesterone, resulting in increased frequency of gonadotropin-releasing hormone (GnRH) pulses. This, in turn, leads to excess luteinizing hormone (LH) secretion.

Increased LH preferentially stimulates theca cell proliferation, which enhances androgen production, while simultaneously suppressing granulosa cell function, impairing estrogen synthesis and leading to follicular arrest a hallmark of anovulatory PCOS.

Molecular Pathways Involved

At the molecular level, androgens reduce the expression of progesterone receptors, weakening the regulatory hormonal feedback essential for normal reproductive function. Furthermore, androgens may modulate GABA_A receptor activity, which affects GnRH neuron sensitivity, contributing to neuroendocrine imbalance. Additionally, the down regulation of hepatic nuclear factor-4α (HNF-4α) suppresses the production of sex hormone-binding globulin (SHBG), increasing the availability of circulating free androgens and intensifying clinical symptoms.

Metabolic and Inflammatory Impacts

Hyperandrogenism also contributes to metabolic disturbances**,** primarily by inhibiting the expression of GLUT-4, a key insulin-responsive glucose transporter. This contributes to insulin resistance a common metabolic feature of PCOS. Moreover, HA encourages central adiposity**,** which further aggravates insulin resistance and creates a self-perpetuating cycle.

From an inflammatory standpoint, elevated androgens stimulate pro-inflammatory cytokines, notably interleukin-6 (IL-6), thereby enhancing systemic inflammation. HA is also linked with increased oxidative stress, which exacerbates cellular damage, particularly in tissues involved in glucose metabolism.

Disruptions in cortisol metabolism and Steroidogenesis

Altered cortisol metabolism is another feature of PCOS. Enhanced 5α-reductase activity combined with diminished 11β-hydroxy steroid dehydrogenase type 1 (11β-HSD1) activity accelerates cortisol clearance in peripheral tissues. This disrupts the normal feedback inhibition on adrenocorticotropic hormone (ACTH)**,** resulting in increased adrenal androgen synthesis.

In addition, genetic variants**,** especially mutations in CYP genes involved in steroid biosynthesis, have been associated

with dysregulated androgen production in women with PCOS [1, 5].

Insulin resistance and hyperinsulinemia in PCOS

Insulin resistance (IR) is a core metabolic feature of polycystic ovary syndrome (PCOS), characterized by a reduced cellular response to insulin, leading to impaired glucose uptake and elevated blood glucose levels. Despite being commonly associated with obesity, IR is observed in 50-70% of PCOS cases, including those with normal body weight, indicating that insulin dysfunction in PCOS is not solely dependent on adiposity.

As a compensatory mechanism, pancreatic β-cells increase insulin secretion, resulting in hyperinsulinemia, which plays a central role in the pathophysiology of PCOS, influencing various endocrine and metabolic pathways:

1. Ovarian Effects

Excess insulin directly stimulates theca cells in the ovaries to produce androgens. It also up regulates LH receptor expression and works synergistically with Luteinizing Hormone (LH) to enhance CYP17 enzyme activity, promoting testosterone synthesis. This androgen excess disrupts follicular maturation, leading to anovulation and ovarian dysfunction.

2. Hormonal Imbalance

Hyperinsulinemia reduces the production of insulin-like growth factor-binding protein-1 (IGFBP-1), increasing IGF-1 availability, which further drives androgen production. Insulin also suppresses aromatase activity, limiting the conversion of androgens into estrogens and maintaining a hyperandrogenic state. Moreover, insulin decreases sex hormone-binding globulin (SHBG) synthesis in the liver, increasing free testosterone levels in circulation.

3. Impaired Folliculogenesis

The combination of insulin resistance and elevated insulin impairs follicle development, contributing to irregular menstruation**,** anovulation, and subfertility due to the accumulation of immature follicles.

4. Neuroendocrine Disruption

Hyperinsulinemia affects the hypothalamic-pituitary axis by enhancing the secretion of gonadotropin-releasing hormone (GnRH) and LH**,** intensifying the neuroendocrine imbalance seen in PCOS.

5. Metabolic and inflammatory consequences

Insulin resistance contributes to central fat accumulation and elevated free fatty acids, which exacerbate metabolic dysfunction. Chronic hyperglycemia further triggers inflammatory pathways, including increased TNF-α production, which can perpetuate insulin resistance and promote systemic inflammation.

Over time, persistent insulin resistance and hyperinsulinemia may lead to β-cell dysfunction, decreasing insulin production and increasing the risk of type 2 diabetes.

Insulin resistance and hyperinsulinemia are central contributors to both the reproductive and metabolic disturbances observed in PCOS. Their role in promoting ovarian androgen excess**,** hormonal imbalance, and chronic inflammation underpins many of the clinical manifestations of the syndrome, including infertility, menstrual irregularities, and metabolic syndrome. Therefore, targeting insulin resistance is a critical component of effective PCOS management and in the prevention of long-term complications such as type 2 diabetes [5, 6].

Oxidative Stress and Its Role in PCOS Pathophysiology

Oxidative stress (OS) arises from an imbalance between reactive oxygen species (ROS) and the body's antioxidant defenses. Key ROS molecules such as superoxide (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radicals (OH^-) along with reactive nitrogen species (RNS), play important physiological roles in cell signalling, growth, differentiation, and steroid hormone production.

In polycystic ovary syndrome (PCOS), elevated oxidative stress activates the NF-κB signalling pathway, triggering the release of pro-inflammatory cytokines like TNF-α and IL-6, both of which are implicated in the development of insulin resistance (IR).

Excessive ROS and RNS can also directly interfere with insulin signalling pathways by altering protein phosphorylation, disrupting insulin receptor activity, and thereby intensifying insulin resistance. Moreover, oxidative stress contributes to adipocyte hypertrophy and fat accumulation, further compounding obesity-related metabolic disturbances seen in PCOS [7].

Gut dysbiosis and mitochondrial dysfunction in PCOS

The gut micro biome, made up of a vast array of microorganisms including bacteria, fungi, archaea, and viruses, plays a vital role in regulating digestion, metabolism, immune function, and overall homeostasis. Often described as the body's "second genome," this microbial ecosystem influences systemic health, and its imbalance termed gut dysbiosis—has been linked to multiple chronic disorders, including polycystic ovary syndrome (PCOS).

Evidence from both clinical studies and animal models indicates that individuals with PCOS often experience distinct shifts in gut microbial composition. While the overall richness (alpha diversity) of the micro biome may remain constant, beta diversity reflecting differences in microbial profiles between individuals is frequently altered. For example, DHEA-induced PCOS models in rodents show significant declines in beneficial gut bacteria such as *Turicibacter*, *Anaerofustis*, and *Clostridium sensu stricto*, suggesting a correlation between gut health and PCOS development.

The DOGMA hypothesis (Dysbiosis of Gut Microbiota Theory) posits that dietary and environmental factors disrupt gut microbiota, increasing intestinal permeability. This allows harmful molecules like lipopolysaccharide (LPS) and zonulin to enter the bloodstream, triggering chronic inflammation via the TLR-4/NF-κB pathway. This inflammatory state contributes to insulin resistance and excess androgen production, both central features of PCOS.

Moreover, androgen excess itself can further alter gut microbial balance, creating a feedback loop that exacerbates hormonal disturbances. Faecal microbiota transplants (FMT) from androgen-exposed mice to germ-free mice have replicated metabolic, endocrine, and reproductive abnormalities, reinforcing the micro biome's key role in PCOS.

Increased levels of Akkermansia and sulphur-reducing bacteria in PCOS models have been linked to higher LPS and interferon-gamma (IFN-γ) levels. IFN-γ promotes pyroptosis in ovarian macrophages, leading to disrupted estrogen synthesis and apoptosis of granulosa cells—both of which contribute to ovarian dysfunction. Interventions with

metformin and disulfiram have shown promise in restoring microbial balance and reducing inflammation.

Recent studies have also highlighted the role of Bacteroides vulgatus in altering bile acid metabolism notably affecting glycodeoxycholic acid (GDCA) and tauroursodeoxycholic acid (TUDCA) which intensifies inflammation through reduced production of the anti-inflammatory cytokine IL-22. Restoring IL-22 levels has been shown to improve insulin sensitivity, ovarian function, and fertility.

Additionally, *B. vulgatus* may elevate agmatine, a metabolite that activates the farnesoid X receptor (FXR), reducing GLP-1 secretion and amplifying pro-inflammatory responses via cytokines such as IL-6, IL-8, IL-1β, and IL-18. These mechanisms further compound metabolic and reproductive disruptions in PCOS [4].

Polycystic Ovary Syndrome (PCOS) is primarily characterized by hormonal dysregulation, chronic low-grade inflammation, insulin resistance, and elevated androgen levels. These interrelated factors disrupt normal follicular development and increase the risk of long-term complications such as type 2 diabetes and endometrial carcinoma.

Current international diagnostic guidelines highlight three core criteria for PCOS diagnosis: clinical or biochemical hyperandrogenism, polycystic ovarian morphology, and chronic anovulation. Additionally, a variety of environmental influences-including geographical location, nutritional habits, socioeconomic conditions, and exposure to environmental toxins-are believed to contribute to both the onset and progression of PCOS, as well as influence treatment outcomes [1].

Diagnosis and management of polycystic ovary syndrome (PCOS)

Diagnostic Criteria

Recent international consensus, building on the Rotterdam criteria, recommends diagnosing PCOS in adult women based on at least two of the following:

1. Clinical or biochemical evidence of hyperandrogenism
2. Irregular or absent ovulation
3. Polycystic ovarian morphology (PCOM) detected via ultrasound or elevated anti-Mullerian hormone (AMH) levels

When both hyperandrogenism and ovulatory dysfunction are present common in about 70% of cases additional imaging or AMH assessment is unnecessary. However, in women presenting with only one criterion, further evaluation using ultrasound or AMH testing is warranted. Biochemical testing should be considered only if clinical signs of hyperandrogenism are absent.

In adolescents, both hyperandrogenism and ovulatory dysfunction must be present for diagnosis. Due to the overlap with normal pubertal changes, AMH and ultrasound are not advised during adolescence. Postmenopausal diagnosis relies on retrospective assessment of prior symptoms, given the lack of ongoing diagnostic features.

Health Risks and Comorbidities

PCOS is associated with increased risks for metabolic and cardiovascular conditions including insulin resistance, type 2 diabetes, metabolic syndrome, endometrial cancer, and obstructive sleep apnea regardless of body weight. First-degree relatives of affected individuals should also undergo cardiometabolic risk screening. During pregnancy, PCOS increases the likelihood of miscarriage, gestational diabetes, hypertensive disorders, preeclampsia, preterm delivery, and caesarean section, warranting early screening and intervention in the preconception or antenatal period.

Adolescent Considerations

Diagnosing PCOS in adolescents requires a cautious approach due to physiological changes during puberty that can mimic the syndrome. Adolescents showing only one characteristic (e.g., hyperandrogenism or irregular menses) are considered "at risk" and should be followed over time. These individuals often exhibit intermediate hormonal or metabolic abnormalities. Timely identification allows for earlier intervention and avoids masking symptoms with hormonal therapy. Treatment may include metformin or combined oral contraceptives, depending on individual needs.

Psychological Support

Mental health concerns including anxiety, depression, disordered eating, and poor self-image are prevalent in PCOS and may hinder treatment adherence. Routine psychological screening is recommended, with therapies such as cognitive behavioural therapy (CBT) tailored to individual preferences. Despite the high burden, mental health issues in PCOS are frequently overlooked, underscoring the need for empathetic and stigma-free support.

Integrated Care Models

Management of PCOS benefits from a multidisciplinary, patient-centred approach. Informed decision-making and access to reliable, evidence-based resources improve both clinical outcomes and patient satisfaction. Training healthcare providers in PCOS-specific care and establishing integrated services like the Victorian PCOS model in Australia are critical to addressing the diverse needs of affected individuals.

Lifestyle and Pharmacologic Treatment

Lifestyle intervention is the cornerstone of PCOS management. Nutritional strategies and regular physical activity are beneficial even without significant weight loss. No universal diet is recommended; instead, personalized approaches such as the DASH or Mediterranean diets based on patient preferences are encouraged. Behavioural support further enhances adherence and outcomes.

For pharmacologic management:

- **COCPs:** Are the first-line treatment for menstrual regulation and reducing androgenic symptoms, especially those with ≤30 µg estrogen.
- **Metformin:** Is recommended for patients with BMI ≥25 kg/m² to improve insulin resistance, glucose metabolism, and menstrual function.
- **Anti-androgens:** May be considered for hirsutism unresponsive to COCs, though their use requires contraception due to teratogenic potential.
- **Inositol supplements:** Are an option based on patient values and preferences, though their effectiveness in ovulation induction and weight management remains inconsistent [8].

Principles of pharmacological management in PCOS

Effective treatment of PCOS should be patient-centred, tailored to the individual's symptoms, goals, and preferences. Ongoing assessment is essential to monitor efficacy, side effects, and to adjust therapy as needed. Clinicians must avoid weight bias and provide respectful care regardless of body size.

Treatment is prioritized based on primary concerns such as menstrual irregularity, metabolic disturbances, hyperandrogenism, or fertility. Many individuals may conceive naturally or with minimal intervention; therefore, lifestyle and metabolic optimization should precede pharmacotherapy aimed at fertility. For those not planning pregnancy, contraception is recommended.

1. Combined Oral Contraceptive Pills (COCPs)

COCPs are the first-line option for addressing menstrual irregularities and symptoms of hyperandrogenism like acne and hirsutism. Their key effects include:

- Reducing ovarian androgen production
- Increasing hepatic SHBG to lower free androgen levels
- Utilizing newer progestins with antiandrogenic properties

COCPs should be prescribed only for individuals with clear menstrual or androgenic issues, not routinely for all PCOS cases. Low-dose estrogen formulations ($\leq$ 30 µg) are preferred. Preparations containing cyproterone acetate are generally avoided due to increased risk of thromboembolism. Progestogen-only pills, while protective for the endometrium, do not significantly improve hyperandrogenic symptoms.

2. Metformin

Although off-label for PCOS, metformin is widely used to manage metabolic dysfunction by improving insulin sensitivity and decreasing hepatic glucose output. Its benefits include:

- Decreases in weight, BMI, and waist-hip ratio
- Better glycemic and lipid profiles

These improvements are most notable in individuals with BMI $\geq$ 25 kg/m² and when combined with lifestyle interventions. Metformin may also restore ovulation and regular menstrual cycles, making it a viable option for those desiring fertility. It may reduce gestational weight gain and preterm birth, though its safety and efficacy during pregnancy remain under investigation.

3. Inositol Supplements

Over-the-counter inositol acts as a mild insulin sensitizer. While a 2024 meta-analysis found slight improvements in fasting insulin and insulin sensitivity, clinical effects on ovulation, weight, and hirsutism were limited. Inositol is better tolerated than metformin but generally offers inferior outcomes, particularly regarding central adiposity and androgenic features. Due to dosage uncertainty, its use should involve shared decision-making.

4. Anti-Obesity Medications

Weight-loss agents like orlistat and GLP-1 receptor agonists (e.g., liraglutide, semaglutide) may be considered when obesity coexists with PCOS, following broader obesity treatment guidelines. These should supplement lifestyle changes. Contraception is essential if using GLP-1 agonists due to limited pregnancy data. Common side effects include gastrointestinal upset, and weight regain frequently occurs upon discontinuation. These medications are not recommended for adolescents due to inadequate paediatric data.

5. Antiandrogens

Antiandrogen agents are used for persistent hyperandrogenic symptoms when COCPs or cosmetic interventions prove ineffective after six months, or when COCPs are contraindicated.

Mechanisms include

Blocking androgen receptors, curtailing androgen synthesis, inhibiting 5-α-reductase Spironolactone is the preferred agent due to fewer side effects. Alternatives like finasteride, flutamide, and bicalutamide pose increased hepatotoxicity, and prolonged use of cyproterone acetate has been linked to a heightened meningioma risk. Antiandrogens are teratogenic and must be paired with reliable contraception. In cases of female-pattern hair loss, combining antiandrogens with COCPs may improve psychological well-being and hair outcomes [9].

Research Methodology

Given the growing interest in herbal medicine for the management and prevention of polycystic ovary syndrome (PCOS), this review explores the mechanisms of action, bioactive compounds, and therapeutic potential of selected medicinal plants used in PCOS treatment. Relevant literature was collected from reputable scientific databases, including PubMed, Google Scholar, Scopus, Cross Ref, and Hinari**.** The following sections provide a comprehensive analysis of specific herbal remedies, highlighting their effectiveness against PCOS through various biological pathways [3].

Limitations and Adverse Effects of Conventional Pharmacotherapy in PCOS

While pharmacological treatments are widely used in managing polycystic ovary syndrome (PCOS), their long-term use is often accompanied by notable adverse effects. Anti-androgens such as flutamide have shown efficacy but pose risks of hepatotoxicity, particularly at higher doses or extended durations. For instance, a case involving a teenage girl who received 500 mg/day of flutamide for facial hirsutism resulted in acute liver failure, highlighting the drug's potential severity.

Oral contraceptive pills (OCPs), another commonly used therapy to regulate menstrual cycles and reduce hyperandrogenic symptoms, are associated with an elevated risk of venous thromboembolism (VTE), especially in patients requiring prolonged treatment or with pre-existing cardiovascular risk factors.

The combination of metformin and clomiphene citrate**,** typically used to induce ovulation in women with infertility, has shown significant effectiveness but also a high incidence of gastrointestinal and systemic side effects**.** In a clinical trial of 626 women undergoing this combination therapy for six months, many participants discontinued due to adverse reactions.

Reported side effects of clomiphene citrate included

Nausea (39%), abdominal pain or discomfort (53%), Headache (44%), Diarrhoea (23%), Mood disturbances (15%), Bloating or gas (18%), Hot flushes (28%)

Common adverse effects of metformin

Gastrointestinal discomfort (59%), Vomiting (30%), Headaches (42%), Nausea (62%), Diarrhoea (65%), Mood changes (17%), Flatulence (18%).

There has even been a fatal case linked to metformin, underlining the importance of cautious prescribing and individualized treatment planning.

In general, synthetic drugs such as metformin**,** used to address insulin resistance and hyperandrogenism-related anovulation, can lead to digestive issues and poor treatment adherence. This necessitates a careful balance between therapeutic benefit and potential harm, with close patient monitoring throughout the treatment course [2, 10].

Concept and application of plant based therapies in PCOS

In recent years, there has been a renewed global interest in herbal medicine, largely driven by the adverse effects, limited efficacy, and growing resistance associated with many modern synthetic drugs. Traditional remedies offer a promising alternative, especially in the management of chronic conditions. It is estimated that nearly 75% of therapeutic agents used globally have origins in traditional or folk medicine. In India, around 70% of modern pharmacological compounds are either derived directly from natural sources or are chemically modified versions of plant-based prototypes. According to the World Health Organization (WHO), over 80% of the population in developing countries relies on herbal medicine to meet their primary healthcare needs. Herbal treatments encompass raw herbs, plant parts, processed herbal materials, finished formulations, and active phytochemicals. Systems such as Ayurveda, Unani, Siddha, and other branches of AYUSH in India have preserved the use of medicinal plants for centuries [11, 12, 13].

Role of herbal medicine in PCOS Management

Polycystic Ovary Syndrome (PCOS) is becoming increasingly common among Indian women, with prevalence estimates ranging from 20% to 25% in the reproductive age group. A growing number of adolescents are also being diagnosed. Although pharmacological agents such as clomiphene citrate, metformin, spironolactone, nafarelin, and troglitazone remain standard treatments, long-term use often leads to side effects such as gastrointestinal disturbances, menstrual irregularities, and metabolic issues like insulin resistance and weight gain.

These challenges have contributed to a shift toward more natural treatment modalities. Herbal medicine, along with lifestyle changes, yoga, and naturopathy, is gaining popularity as a safer and more sustainable approach. Herbal therapies are particularly advantageous in managing chronic disorders like PCOS due to their lower risk of side effects and better suitability for prolonged use.

Traditional medical systems including Ayurveda, Unani, and Traditional Chinese Medicine have long utilized medicinal plants to address gynaecological issues. Many of these botanicals possess bioactive compounds that help correct hormonal imbalances, enhance insulin sensitivity, and exert anti-inflammatory effects, thereby targeting multiple aspects of PCOS pathophysiology [10].

Table 1: Herbal treatments for symptoms and complications of PCOS [2]

Symptom/Complication	Herb Used for Treatment
Hormonal Imbalance	Orchid Tree
Antioxidant Support	Cinnamon
Hormonal Regulation	Amla, Haritaki
Depression & Mood Swings	St. John's Wort
Reproductive Dysfunction	Gokharu
Menorrhagia	Guggle
Regulation of Menstrual Flow	Guduchi
Cholesterol Management	Black Seed

Promising Herbal Remedies for PCOS

Several herbs have shown potential therapeutic benefits for managing PCOS, offering natural alternatives to conventional treatments, these include:

- Aloe Vera, Cinnamon, Fenugreek, and Liquorice: Known for improving blood glucose control and insulin resistance.
- Chamomile, Green Tea (*Camellia sinensis*), Spearmint (*Mentha spicata*), and Silymarin: Beneficial for ovarian health and antioxidant activity.
- Fennel (*Foeniculum vulgare*) and *Heracleum persicum*: Help regulate menstrual cycles and maintain hormonal balance.
- Red Onion: Demonstrates cholesterol-lowering effects in women with PCOS.
- Nigella sativa: Supports menstrual cycle regulation.

Supporting Clinical Findings

- Cinnamon enhances menstrual regularity, improves lipid profiles, and boosts antioxidant levels.
- Green Tea consumption in overweight women with PCOS results in weight reduction and better insulin regulation.
- Spearmint has proven effective in reducing hirsutism.
- Satapushpa and Shatavari (Ayurvedic herbs) have potential in reducing ovarian volume.
- Fennel Seed Infusions combined with Dry Cupping Therapy effectively treat oligomenorrhea.
- Furocyst, an extract of Fenugreek, has significantly improved PCOS symptoms in clinical trials.

These findings support the inclusion of evidence-based herbal treatments as part of a comprehensive PCOS management plan. When combined with dietary changes and practices like yoga, these natural remedies offer a lower-risk, holistic alternative to conventional pharmacotherapy. The development of polyherbal formulations may further reduce treatment duration, minimize side effects, and decrease costs, making them a practical choice for long-term PCOS management [10].

Table 2: Some Plants used for PCOS

Scientific Name	Family	Part used	C.C.	Medicinal Activity	Ref.
Aloe barbadensis (Aloe Vera)	Liliaceae	Leaves (hydro alcoholic extract)	Aloe emodin, Barbaloin	It suppresses ovarian androgen-producing enzymes (3β-HSD, 17β-HSD), reduces ovarian size and cholesterol synthesis, boosts estrogen production, restores glucose sensitivity, and improves lipid profiles. Also associated with decreased progesterone levels.	[3, 14]
Chamomilla matricaria (Chamomile)	Asteraceae	Flower (alcoholic extract)	Apigenin, Gallic acid, Matricin Farnesene, coumarin derivatives, Kamazelin,	It improves ovarian function, increases follicle development, and enhances LH secretion.	[14]
Vitex agnuscastus (Chaste Tree)	Verbenaceae	Fruits, Leaves, and Woody	iridoids, flavonoids, steroids, alkaloids, glycosides,	They elevate serum progesterone, lower testosterone by enhancing aromatase activity, and possess anti-inflammatory and estrogen-	[14]

		parts		modulating properties.	
Cinnamomum zeylanicum (Cinnamon)	Lauraceae	Bark	Cinnamaldehyde and Eugenol	They have antioxidant activity, improves menstrual regularity, enhances lipid profiles, and supports ovulation.	[2]
Panax ginseng (Ginseng)	Araliaceae	Root	Ginsenoside Rb1, Rb2, Rc, Rd, Re, Ro and Ra	Constituents (ginsenosides Rb1, Rg1, etc.) exhibit estrogen-like activity, increasing estradiol and modulating gonadotropins. Supports fertility and relieves menopausal symptoms.	[3, 14]
Stachys lavandulifolia (Stachys)	Lamiaceae		Flavonoid-Apigenin, Saponins, Quinine, Iridoids, Phenolic acids	Exhibits estrogenic effects through receptor interaction, helping modulate estrogen activity.	[14]
Foeniculum vulgare (Fennel)	Apiaceae	Dried ripe Fruits	Trans Anethole, Tocopherols-α-tocopherol, β-tocopherol, γ-tocopherol and δ-tocopherol, Ascorbic acid, Dianethole, Photoanethole, Estrogole, Fenchine, and p-Anisaldehyde	Promotes progesterone production, improves endometrial thickness, reduces estrogen-induced uterine changes, and supports healthy uterine tissue.	[3, 14]
Glycyrrhiza glabra L (Licorice)	Fabaceae	Roots (hydro alcoholic)	Glycyrrhizin and glycyrrhizin acid, flavonoids such as glabridin and liquiritin, isoliquiritin, glabrene	Exerts anti-androgen effects, supports estrogen biosynthesis, inhibits 11β-HSD2, and improves insulin-mediated glucose regulation.	[14]
Vitex agnus castus (chaste berry, Nirgundi)	Verbenaceae	Root, leaves, flowers, fruits, bark	Viteagnusin and Flavonoids including Apigenin, 3-Methylkaempferol, Luteolin and Casticin, Bornyl acetate, Limonene, 1,8-Cineol, α-Pinene and β-Pinene Viteagnusin, Vitexilactone, Rotundifuran and Vitex lactam A, Chrysoplenetin and Chrysosplenol D and Iridoids such as Cynaroside		[3, 15]
Curcuma longa (Turmeric)	Zingeberaceae	Root	Curcumins, Curcuminoids, Ferulic acid, Eugenol, Ascorbic acid, Vanillic acid, Caffeic acid, Syringic acid, Protocatechuic acid, and p-coumaric acid, and Terpenoids-Turmerone, α-Turmerone, Camphene, β-Sesquiphellandrene, γ-Terpinene and Carotene	Reduce progesterone while elevating estradiol, improving follicular development, corpus luteum formation, and promoting antioxidant, hypoglycaemic, and lipid-lowering effects.	[3]
Linum usitatissimum (linseed)	Linaceae	Seed	Lignans-Secoisolariciresinol and Secoisolariciresinol diglycoside-SDG, and Linolenic acid, Omega-3 fatty acids	Regulates estrogen, lowers testosterone and free androgen levels, reduces BMI and insulin, and decreases hirsutism.	[3, 14]
Mentha spicata (Spearmint)	Labiatae	Leaves	Lutein, Rosmarinic acid, Caffeic acid, Salvianolic, Dehydro-salvianolic acid	It have potent antioxidant effects. They reduce free testosterone, improve LH: FSH balance, decrease hirsutism, and shift ovarian morphology toward healthy follicular dynamics.	[3]
Cocos nucifera (Coconut)	Arecaceae	roots	Lupeol methyl ether, Skimmiwallin and Isoskimmiwallin	It show anti-androgenic activity, modulate gonadotropin levels, restrain ovarian size, and support uterine health.	[3]
Punica granatum (Pomegranate)	Punicaceae	seed extract	Quercetin, Gallocatechin, Catechin, Epicatechin, Kaempferol	Shown to normalize testosterone, estrogen, and androstenedione in women consuming it regularly.	[3]
Cimicifuga racemosa (black cohosh)	Ranunculaceae	Root or Rhizome	Hydroxycinnamic acids, Ferulic acid, Isoferulic acid and Caffeic acid, Cimicifugic acids (Glycolyl Phenylpropanoids)	Acts via estrogen receptor modulation in the pituitary, reducing LH output and ovarian cyst development via flavonoid activity.	[3]
Pimpinella anisum L **(**Anise)	Apiaceae	Seed	Trans-anethole, Anisketone, Anisaldehyde and Methyl Chavicol.	Supports menstrual regularity by regulating LH: FSH secretion and relieving oligomenorrhea.	[3]
Trigonella foenum-graecum (Fenugreek)	Fabaceae	Seed	Terpenoids-β-pinene, β-caryophyllene, Camphor and Neryl acetate	Help suppress ovarian cyst formation and improve hormonal and ovarian function.	[3]
Zingiber officinalis (Ginger)	Zingeberaceae	Rhizome	Gingerol and Shogaol	Inhibit prostaglandin synthesis, help balance estrogen-progesterone, and regulate androgen and reproductive hormones.	[3]
Tribulus terrestris (Gokharu)	Zygophyllaceae	Seed	Furostanol, Spirostanol, Tigogenin, Diosgenin, Hecogenin, Neohecogenin, Chlorogenin, Kaempferol, Kaempferol-3-glucoside, kaempferol-3-Rutinoside and Tribuloside	Steroidal saponins and flavonoids normalize estrous cycles, hormonal balance, and stimulate ovarian follicles, serving as a fertility tonic.	[3]
Asparagus racemosus (Shatavari)	Asparagaceae	Root	Shatavarin, Asparagamine A, Flavonids-Kaemperol, Rutin	Prevent cyst formation and disease recurrence, enhancing long-term ovarian health.	[15, 16]
Camellia sinensis (Green Tea)	Theaceae	Leaves	Caffeine, Tannins, and Polyphenols	Reduce LH levels, body and ovarian weight, improve insulin resistance, and beneficially alter follicular structure.	[17, 18, 19]
Withania somnifera (Ashwagandha)	Solanaceae	Root	Withanolides and Flavonoids Chlorogenic acid, Withanone	Help lower androgen levels, supporting hormonal equilibrium.	[15, 16]

Conclusion

Polycystic Ovary Syndrome (PCOS) is one of the most common reproductive disorders, primarily caused by hormonal imbalances in women. It is particularly prevalent in developing countries, such as India, where socioeconomic and physical conditions contribute to its higher incidence. Healthy menstruation is essential for overall health and fertility in women. Although numerous synthetic and chemical treatments for PCOS exist, long-term use of these drugs is often linked to side effects such as constipation, heartburn, nausea, gastric discomfort, and diarrhoea. Moreover, these medications are not affordable for many, especially in developing regions like Southeast Asia and Africa, where access to high-quality drugs is limited.

Given these challenges, there has been a resurgence in the use of herbal products as alternatives to modern drugs, largely due to their fewer side effects, interactions, and the failure of conventional treatments for chronic diseases. According to the World Health Organization (WHO), at least 80% of people in developing countries rely on herbal remedies. In traditional Indian medicine, numerous plants are utilized due to their minimal side effects and lower risk of interactions. These plants are integral to systems like Ayurveda, Siddha, Unani, and AYUSH. Herbal medicines typically include herbs, plant parts (e.g., leaves, flowers, roots, seeds), and finished herbal

products that are easily accessible and can even be grown in home gardens.

The aim of this review is to explore the latest advances in plant-based treatments for PCOS. It focuses on the pharmacological properties, benefits, and potential side effects of various plants, their use in regular diet plans, and the supporting documentation from sources like Google Scholar, previous review papers, books, and guidelines. This review will serve as a valuable resource for researchers developing effective and affordable PCOS treatments.

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