



# A Review Article On Yhe Treatment Of Polycystic Ovarian Syndrome (Pcos) By Herbal Drugs

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## Abstract

Polycystic Ovarian Syndrome (PCOS)-related infertility is a global issue that is becoming more prevalent these days. There are over 5 million of them, or nearly 7% of all adult women. Women in the reproductive age range are typically affected by the illness. It is characterized by polycystic ovaries, persistent anovulation, and hyperandrogenism, which can result in infertility, hirsutism, acne, and irregular menstrual cycles. There are numerous pharmaceutical treatments available for polycystic ovarian syndrome. They have a number of adverse effects. These days, ginseng, tulsi, amla, flax seeds, cinnamon, chaste berries, fennel seeds, liquorice, curcumin, aloevera, and ginseng are among the herbal medications used to treat polycystic ovarian syndrome. Hormonal balance, decreased hirsutism, increased metabolic rate, and higher insulin sensitivity are only a few of its advantages versus negative effects. Women with PCOS benefited from the herbal plant without experiencing any negative side effects. Understanding the natural plants that can be used to treat diseases naturally is the goal of this review. You can use these herbs separately or in combination.

**Keywords:** Polycystic ovarian syndrome, hyperandrogenism, infertility, herbal treatment.

## 1. Introduction

### PCOS (Polycystic ovarian syndrome)

PCOS is a diverse illness that affects women of childbearing age, according to the National Institutes of Health Office of Disease Prevention. There are over 5 million of them, or nearly 7% of all adult women. According to research, PCOS is the most prevalent endocrine condition, affecting 5–10% of girls between the ages of 18 and 44 [1]. PCOS, which was initially identified by Stein and Leventhal in 1953, is linked to polycystic ovarian morphology, chronic oligoanovulation, psychological impairment, and metabolic abnormalities, primarily insulin resistance and compensatory hyperinsulinemia, which may be the cause of changes in androgen production and metabolism in reproductive age. Many find it difficult to identify this widespread condition due to a lack of clear diagnostic criteria[2].

The presence of two or more of the following characteristics—chronic oligo-ovulation or anovulation, androgen excess, and polycystic ovaries—defines the clinical diagnosis of polycystic ovarian syndrome [3]. One neuroendocrine characteristic of PCOS is elevated luteinizing hormone secretion frequency, elevated luteinizing hormone serum concentration, elevated amplitude, and elevated LH/FSH ratio [4].

In addition, a number of genetic and environmental variables work together to cause PCOS. Women who are obese, have a family history of PCOS, and exercise little or not at all are more likely to have it. The existence of ovarian cysts, the absence of ovulation, and elevated testosterone levels are among the

findings utilized to treat the illness. The presence of any cysts can be found using ultrasound techniques. The diagnosis can also be made using a number of other factors that produce comparable symptoms, such as hyperplasia, hypothyroidism, and increased blood prolactin levels [5].

According to recent research, there is currently little to no cure for PCOS. The only way to complete the treatment is to make minor lifestyle adjustments, including exercising and losing weight. Birth control tablets are one medication that can help preserve the regularity of the menstrual cycle [6]. Pharmacological interventions include substances that reduce insulin, certain anti-androgens, and progestin-oestrogens together. Due to their high cost, this type of treatment has a number of issues and adverse effects. Weight gain, irregular menstruation, gastrointestinal issues, and insulin resistance are possible side effects [7].

## 2.Pathophysiology of PCOS

Luteinizing hormone (LH) and follicle stimulating hormone (FSH) are the released or produced gonadotropic releasing hormones. Normal follicular growth requires less intraovarian androgen. Follicle formation is facilitated by FSH and the stimulating aromatase enzyme, which encourages the conversion of testosterone to estrogen in granulosa cells. LH stimulates the development of theca cells, which starts the maturation of oocytes.

PCOS raises LH levels, while lowering FSH levels results in increased androgen production and decreased aromatase enzyme levels with immature follicle formation. Abdominal obesity is linked to excessive testosterone in PCOS, which causes dyslipidemia and hyperinsulinemia. Hyperinsulinemia decreases sex hormone binding globulin (SHBG), which raises the amount of testosterone in the blood and enhances the production of androgen by theca cells [8].

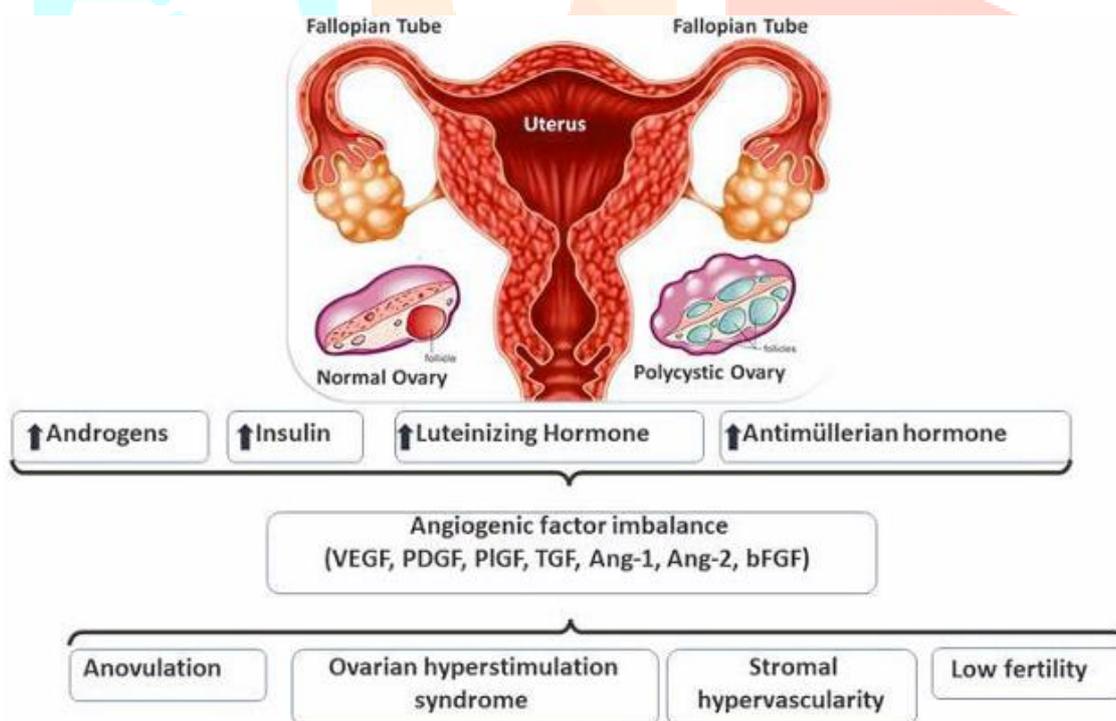


Figure 1: Pathophysiology of PCOS [8]

## 3.Causes

- Genetic predisposition
- Strong stimulation in adrenals in childhood
- Raised insulin levels
- Contraceptive pills
- Hormonal imbalance
- Stress[9]

## 4. Symptoms

Later in your twenties or thirties, symptoms like the onset of your first period or even health problems began. Although each woman experiences these symptoms differently, at least two of the following conditions must exist in order to be diagnosed with polycystic ovarian syndrome:

- Absent periods
- Acne not associated with puberty
- Darkening of the skin
- Excessive hair growth
- Fatigue
- Fluid retention
- Heavy or prolonged periods Infertility
- Irregular periods
- Male pattern baldness
- Menstrual pain
- Mood swings
- Ovarian cysts
- Weight gain[10]

## 5. Organs involved in PCOS:

- OVARY- The female gonad organ present at either side of uterus.
- ADRENAL GLAND- The gland which are placed just above the both the kidneys
- PANCREAS- Gland that produces insulin in our body.
- PITUITARY GLAND- The gland just below the brain, which is responsible for all the Hormonal control.
- UTERUS- Irregular menstrual cycles, endometrial hyperplasia.
- FALLOPIAN TUBE- Increased risk of tubal damage and infertility[11].

## 6. Classification Based on Phenotype

When PCOS is present, four distinct phenotypes can be identified: A, B, C, and D. Overall, it seems that the degree of monthly irregularity, body mass index, and hyperandrogenism (HA) are independent predictors of metabolic dysfunction, but not ovarian morphology [12,13].

- PCOS is classified into four phenotypes as:

(A) By ultrasound, numerous polycystic ovaries, oligoanovulation, and hyperandrogenism. (B) By ultrasound, normal appearance of ovaries, oligoanovulation, and hyperandrogenism. (C) By ultrasound, polycystic ovaries with normal routine menses and hyperandrogenism. (D) By ultrasound, polycystic ovaries, oligoanovulation, without hyperandrogenism.

### 1. PCOS Phenotypes A and B

Classic PCOS is the term used to describe PCOS phenotypes A and B. Menstrual dysfunction is more severe, insulin secretion is higher, insulin resistance is higher, and the risk of metabolic syndrome is higher in women with the classic PCOS phenotypes A and B. Compared to other PCOS phenotypes, classic PCOS has a higher prevalence of obesity and atherogenic dyslipidemia (AD) [14]. Compared to normal healthy controls and other PCOS phenotypes with normal androgen, PCOS phenotypes A and B also have a higher incidence of hepatic steatosis [15]. A markedly high amount of anti-mullerian hormone is another characteristic of classic PCOS [16].

### 2. Phenotype C – Ovulatory PCOS

Compared to individuals with classic and non-hyperandrogenic PCOS, phenotypic C (ovulatory PCOS) patients frequently have somewhat raised serum insulin, atherogenic lipids, and androgen levels, as well as high hirsutism ratings. Compared to other forms of PCOS, metabolic disorders are also prevalent in

ovulatory PCOS [17]. Higher socioeconomic level was associated with the ovulatory phenotype in a population of PCOS patients from Italy [18]. The variation in ovulation patterns in high socioeconomic groups may be partially explained by the distribution of tissue fat and insulin levels brought on by dietary choices.

### 3. Phenotype D – Nonhyperandrogenic PCOS

In contrast to healthy controls, phenotype D is characterized by the lowest metabolic dysfunction [19,20], normal androgen, and modestly higher levels of other endocrine variables [21,22]. In contrast to people with classic PCOS, the endocrine findings include a lower LH/FSH ratio, a low level of T3 and T4, and an enhanced amount of sex hormone-binding globulin [23]. Regular menstrual periods with sporadic irregularities are typical for those with PCOS phenotypic D [24].

### Stages of PCOS

1. Mild PCOS: Minimal symptoms, regular menstrual cycles
2. Moderate PCOS: Irregular menstrual cycles, mild hyperandrogenism
3. Severe PCOS: Significant hyperandrogenism, irregular menstrual cycle, polycystic ovaries.

#### • Variations Associated with PCOS

PCOS is a multifactorial disease and is caused by a number of abnormalities. All genes/mutations that affect ovaries directly or indirectly are associated with PCOS.

### I] Genes involved in ovarian and adrenal steroidogenesis

The most common endocrine disorder associated with PCOS is an elevated androgen level. Hence, in uncovering the reason for the elevated level of androgen, several genes have been reported to be associated with PCOS, as follows.

#### • CYP11a

An enzyme that is necessary for the intermediate stage of cholesterol conversion to progesterone is encoded by the CYP11a gene. This stage of the cholesterol conversion process is rate-limiting [25]. Furthermore, in a study of 97 infertile women, Gharani et al. found that variation and polymorphism were linked factors [26]. The results were confirmed by two other investigations conducted in China and Greece, which found CYP11a to be a factor associated with PCOS [27, 28]. The findings were later not replicated by a sizable investigation carried out in the UK [29].

#### • CYP21

An enzyme encoded by CYP21 catalyzes the conversion of 17-hydroxyprogesterone to 11-deoxycortisol during the manufacture of steroid hormones. PCOS is further exacerbated by inadequate steroidogenesis anabolism caused by a less active enzyme as a result of variance. According to Witchel et al., heterozygous CYP21 is linked to hyperandrogenism and PCOS-like symptoms [30]. In 114 PCOS-afflicted women, there was no direct correlation between CYP21 variation and PCOS [31].

#### • CYP17

An enzyme (P450c17 $\alpha$ ) encoded by CYP17 catalyzes the conversion of pregnenolone and progesterone into 17-hydroxypregnenolone and 17-hydroxyprogesterone. According to Rosenfield et al., PCOS patients have higher androgen levels [32]. According to Wickenheisser et al., theca cells exhibit elevated CYP17 expression [33]. A polymorphism in the promoter area linked to PCOS was found by Carey et al. [34].

#### • CYP19

On chromosome number 15q21.2 is the CYP19 gene that produces aromatase p450, which is essential for the synthesis of estrogen [46]. Women with PCOS who are thin or obese have been found to have lower aromatase activity.

### II] Gene involved in steroid hormone effects

#### • Androgen receptor gene

The AR gene, which has 11 exons and codes for a 90 kb tridomain protein, is located on the "q" arm of chromosome X [37]. PCOS is said to be caused by gene mutations and structural disturbance. PCOS results from the interruption of the cellular mechanism caused by "X" chromosomal inactivation, which raises androgen hormone levels. Because the AR gene is found on the X chromosome, disease can be brought on by alterations in just one copy of the gene. Additionally, GWAS revealed that PCOS is caused by a new gene variant [38].

#### • Sex hormone-binding globulin gene

The location of the SHBG gene is on chromosomes 17p13–p12. A 373-amino acid protein is produced by SHBG. By binding to androgens, primarily estrogens and testosterone, the protein product of SHBG regulates the body's levels of sex hormones [39, 40]. Hepatocytes in the liver produce the majority of SHBG. Insulin and androgens are two metabolic variables that regulate the hepatocytes' production of SHBG [41–43]. Females with PCOS have reduced SHBG concentrations, which has been primarily linked to hyperinsulinemia's inhibitory effect on SHBG production [42]. Numerous investigations have reported a substantial correlation between PCOS and a single nucleotide polymorphism in the SHBG gene [44, 45].

### III] Gene involved in gonadotropin action and regulation

#### • Lutein hormone (LH) and its receptor gene

PCOS is commonly attributed to both elevated LH levels and abnormal LH function. These anomalies result in annulations, which in turn lead to PCOS [46]. Consequently, a high quantity of LH increases androgen production [47]. Reduced follicle-stimulating hormone (FSH), which is the negative feedback in response to high LH, may have an indirect effect by aggravating the excess testosterone in the ovaries and decreasing the transfer of androgen to estrogen [48]. To determine whether there may be a connection, researchers have looked at the genes that encode both LH and its receptor in PCOS. First, PCOS patients were found to have a point mutation (Trp8Arg and Ilg15Thr) in a gene that codes for the B subunit [49]. According to reports, 15% of the normal population carries the same mutation, which is nonpathogenic [50]. There have also been reports linking PCOS to polymorphism in the LH  $\beta$ -subunit gene [51].

#### • AMH

The AMH gene can be found at cytogenetic position 13.3 on the long arm of chromosome 19. The gene, which produces a protein implicated in infertility, has five exons [52]. PCOS is linked to variations in the AMH gene. AMH gene variations were identified as powerful predictors of PCOS by whole exome sequencing and GWAS [53].

#### • Follicular stimulating hormone receptor (FSHR)

The 14 exons that make up FSHR are found on chromosome 2's "p" arm. A protein G-coupled receptor, which is encoded by the gene, is necessary for gonad development [54]. A gene mutation causes the structural protein to be disrupted, which results in a hormone imbalance. PCOS is a hormonal imbalance. A higher frequency of the gene was found in the affected persons in north Iraq when polymorphisms in healthy and affected individuals were compared [55].

### IV] Gene involved in insulin action and secretion

#### • The Insulin Gene

Insulin also has a major impact on the androgen synthesis that occurs through receptors on theca cells. Phosphoinositide 3-kinase/protein kinase B is the route that triggers this insulin action, and it becomes active in PCOS theca cells [56]. A high insulin level also promotes the manufacture of androgens, much like LH does [57,58]. Tyrosine hydroxylase and IGF-II are sandwiched by the INS gene at 11p15.5 [59]. A tandem repeat of VNTR occupies the 5 untranslated region [60]. VNTR polymorphism controls the transcriptional rate of INS and IGFII [61]. VNTR can be performed anywhere between 26 and 200 times. PCOS is linked to this VNTR polymorphism [62].

### • INSR

The turmeric protein, which is made up of two alpha and two beta chains, is encoded by this gene [63]. Numerous investigations were carried out in an attempt to determine whether infertility and PCOS were related, however they were unsuccessful [64]. The tyrosine kinase domain-encoding region of INSR in women with PCOS was also investigated by Conway et al. [65]. Women with PCOS had their whole gene scanned by Talbot et al. [66]. There was no correlation between PCOS and ISR in either of these investigations. After a more thorough search of chromosome 19p12.2, D19S884 was shown to have the highest correlation with PCOS [67]. The INSR gene is also located in this chromosomal region.

### • Insulin Receptor Substrate Proteins

Insulin attaches itself to its receptor. Insulin binding causes the receptor to become autophosphorylated. The INS receptor's tyrosine kinase activity then phosphorylates IRS-1 and IRS-2.34. The downstream process subsequently makes additional use of these activated substrates. Numerous investigations have been conducted to determine the relationship between PCOS and the genes of IRS-1 and IRS-2. According to Petermann et al., women with PCOS had a higher frequency of Arg972 IRS-1 [68], however El Mkadem et al. found no discernible difference between PCOS patients and controls [69]. Turkish women with PCOS had a significantly greater frequency of Gly972Arg in IRS-1, according to Dilek et al. [70]. There have been reports of both associations and none at all. These variations draw attention to ethnic and environmental involvement.

### • Calpain10 Gene

CAPN10, which has 12 exons, is found on the long arm of chromosome 2's "q." The heterodimeric protein calcium-dependent cysteine protease is encoded by the gene. Diabetes mellitus type 1 is linked to the gene [71]. Calpain 10 is a protein that disrupts the metabolism and release of insulin. Since PCOS is brought on by low insulin, PCOS is also brought on by a mutation in calpain 10. CAPN10 is therefore a potential gene responsible for PCOS [72].

### V] Other Genes

#### • Fat Mass Obesity (FTO)

Alpha-ketoglutarate is an enzyme that is encoded by the FTO gene. The gene is found on chromosome 16's "q" arm. Obesity and type 2 diabetes have been linked to the gene [73]. One study found that the single nucleotide polymorphism (SNP) rs9939609 was strongly linked to illnesses in Pakistani women with PCOS. Affected women's SNP rs9939609 was noticeably greater than that of the study's healthy participants [74].

#### • PCOS1

PCOS1 is located on chromosome 19p13.2. A number of research have linked the gene to PCOS. The gene, which is actually a susceptibility area on chromosome 19, is also referred to as PCO. In 1971, it was first discovered in two sisters. Urbanek et al. later reproduced the findings in 2005 [75].

#### SRD5A2 and SRD5A1

SRD5A2 is located on chromosome 2p23.1. In 1999, Jakimiuk et al. observed that women with PCOS had higher SRD5A activity than women without PCOS [76]. A variant in SRD5A2 was linked to protection against PCOS, while variants in SRD5A1 were linked to a higher risk of hirsutism and therefore PCOS, according to research conducted later in 2006 on SRD5A2 and SRD5A1 susceptibility to PCOS in hirsutism patients [77].

### VI] Epigenetics of PCOS

Epigenetic alterations are heritable variations in gene expression that are transgenerationally and mitotically heritable but do not result from a change in DNA sequence. Numerous disorders, including PCOS, prostate cancer, and type 2 diabetes, have been linked to epigenetic participation. In models involving rats, monkeys, and sheep, increased androgen secretion throughout fetal life has been linked to illnesses. PCOS has symptoms with several illnesses. Some research have demonstrated that an elevated amount of androgen during fetal life predisposes the offspring to PCOS-like symptoms later on, despite the

ethical restrictions on using humans for testing. Qu and others revealed that granulosa cells' PPARG1 and NCOR1 have differential CPG island methylation, which results in hyperandrogenism-induced epigenetic changes and the emergence of ovarian failure. According to Ning Xu et al., PCOS patients have a different DNA methylation pattern than control groups [78].

## 7. Allopathy medication

Multiple organ systems are impacted by the complex disease known as polycystic ovarian syndrome and pocystic ovarian disorder. The patient's appearance and desire for pregnancy should guide the course of treatment. Birth control tablets are one type of treatment to help regularize periods. drugs and devices used to treat PCOS symptoms and related adverse drug reactions.

Drug	Mechanism	Indication	Dosage
Metformin	Insulin sensitizer	Insulin resistance	500-2000 mg/day
Birth control pills(e.g. Yasmin, Diane)	Hormone regulation	Menstrual irregularities	Varies
Spirolactone	Anti-androgen	Hirsutism, acne	50-100 mg/day
Clomiphene citrate	Ovulation inducer	Infertility	50-100 mg/day
Letrozole	Aromatase inducer	Ovulation induction	2.5-5 mg/day
Gonadotropins	Hormone replacement	Ovulation induction	Varies
Finasteride	5-alpha-reductase inhibitor	Hirsutism	5 mg/day
pioglitazone	Insulin sensitizer	Insulin resistance	15-45 mg/day
Flutamide	Anti-androgen	Hirsutism, acne	250-500 mg/day
Rosiglitazone	Insulin sensitizer	Insulin resistance	4-8 mg/day

**8. Side effects of Allopathy drugs:-** Allopathy drugs used to treat PCOS have various side effects. Here are some common ones [82-86]

### • Hormonal Contraceptives:

#### 1. Birth control pills (e.g., Yasmin, Dianette)

- Weight gain
- Mood swings
- Breast tenderness
- Increased risk of blood clots, stroke
- Headaches
- Nausea

#### 2. Progestin-only pills (e.g., Micronor)

- Irregular bleeding
- Weight gain
- Mood changes
- Breast tenderness

### • Fertility Medications:

#### 1. Clomiphene (Clomid)

- Hot flashes
- Mood swings
- Breast tenderness

- Abdominal pain
- Ovarian cysts
- Multiple pregnancies

## 2. Letrozole (Femara)

- Hot flashes
- Fatigue
- Headaches
- Dizziness
- Nausea

### • Anti-Androgen Medications:

#### 1. Spironolactone (Aldactone)

- Breast tenderness
- Menstrual irregularities
- Fatigue
- Dizziness
- Headaches
- Increased risk of birth defects

#### 2. Flutamide (Eulexin)

- Liver damage
- Diarrhea
- Fatigue
- Headaches
- Dizziness

### • Insulin-Sensitizing Medications:

#### 1. Metformin (Glucophage)

- Nausea
- Diarrhea
- Abdominal pain
- Headaches
- Fatigue
- Lactic acidosis (rare)

#### 2. Pioglitazone (Actos)

- Weight gain
- Edema
- Headaches
- Fatigue
- Increased risk of bladder cancer

## 9. Herbal medication

Polycystic Ovary Syndrome (PCOS) is a hormonal disorder that affects women of reproductive age, causing symptoms like irregular periods, weight gain, acne and infertility. Herbal drug treatment have gained popularity due to their potential benefits and fewer side effects. Here are some herbal remedies that have over benefit PCOS.

Herbal drugs :

1. Improve insulin sensitivity without medications
2. Regulate menstrual cycle naturally
3. Reduce androgen levels without hormonal side effects
4. Enhance fertility without invasive procedures
5. Other a holistic approach to overall well-being

### 1. Liquorice

Biological name: Glycyrrhiza globra

Family: Leguminosae

Chemical constituents: Glycyrrhiza

Use: In the luteal phase of the cycle, nine healthy women aged 22 to 26 had their androgen metabolisms examined in relation to liquorice. For two cycles, they received 3.5 g of a commercial licorice preparation that contained 7.6% W/W glycyrrhizic acid per day. They weren't receiving any other care. Radioimmunoassay was used to evaluate plasma renin activity, serum levels of gonadal and adrenal androgens, cortisol, and aldosterone. Over the course of two months, total serum testosterone steadily dropped. After stopping the treatment, it went back to its pretreatment levels. Probably as a result of blocking 17-hydroxysteroid dehydrogenase and 17-20 lyase, licorice can lower serum testosterone levels. Licorice may be used as an adjuvant treatment for polycystic ovarian syndrome and hirsutism [87].



Fig.Liquorice

## 2. Aloe-Vera

Biological name: Aloe barbadensis

Family: Liliaceae

Chemical constituents: Polysaccharide compounds

Use: The effectiveness of the Aloe vera gel formulation was examined in this study using a PCOS rat model. Letrozole, a nonsteroidal aromatase inhibitor, was given orally to five-month-old Charles Foster female rats in order to cause PCOS. After that, the rats received an oral dose of the Aloe vera gel formulation (1 ml each day for 45 days). Their steroidogenic activity, glucose sensitivity, and estrus cyclicity were all recovered as a result. The development of the PCOS phenotype was stopped by co-treating the aloe vera gel with the inductive substance (letrozole). By restoring the ovarian steroid status and changing important steroidogenic action, the aloe vera gel formulation protects against the PCOS phenotype. The phytochemicals in the extract are responsible for this [88].



Fig. Aloe-Vera

## 3. Flax seed

Biological name: Linum usitatissimum

Family: Linaceae

Chemical constituents: Lignan

Use: This study examined how a 31-year-old woman with PCOS's hormone levels were affected by taking 30g of flaxseed daily. The patient took 83% of the recommended amount of flaxseed over the course of four months. Measurements of height, weight, and fasting blood samples obtained at baseline and at 4-month follow-up showed a significant drop in insulin, total serum testosterone, free serum testosterone, and body mass index (BMI). At the end of the research period, the patient also reported less hirsutism. This case study documented a clinically significant decrease in testosterone levels together with a corresponding decrease in hirsutism [89].



Fig.Flax seed

## 4. Cinnamon

Biological name: Cinnamomum zeylanicum

Family: Lauraceae

Chemical constituents: Phosphatidylinositol 4-kinase

Use: In both in vitro and in vivo experiments, cinnamon extract has been demonstrated to decrease insulin resistance by potentiating the action of insulin via raising phosphatidylinositol 3-kinase activity in the insulin signaling pathway. For eight weeks, fifteen women with PCOS were randomly assigned to either daily oral cinnamon or a placebo. Significant decreases in insulin resistance were observed in the cinnamon group but not in the placebo group when posttreatment insulin sensitivity indices were compared to baseline insulin sensitivity indices using fasting and 2-hour oral glucose tolerance tests [90].



Fig. Cinnamon

### 5. Chaste berry

Biological name: *Vitex agnus-castus*

Family: Verbenaceae

Chemical constituents: Flavonoids, iridoid glycoside, p hydroxybenzoic acid, alkaloids.

Use: This herb is regarded as an adaptogen and has been used for generations to treat hormone imbalances. One of the most popular herbs used to treat PCOS is chaste berry, which helps to regulate and promote pituitary gland function. Luteinizing hormone, which is released by the pituitary gland, can raise progesterone levels while lowering estrogen and androgen levels.



Fig.Chaste berry

### 6. Fennel seeds

Biological name: *Foeniculum vulgare*

Family: Apiaceae

Chemical constituents: Trans anethole-photoanethole, estragole, fenchine, and P- anisaldehyde Dianethole, cinnamic acid, fumaric acid, benzoic acid, vanillic acid.

Use: Due to its anti-hirsutism qualities and ability to lower levels of androgen (male hormones), fennel seeds are thought to benefit in the treatment of PCOS [91].



Fig.Fennel seeds

### 7. Amla

Biological name: *Phyllanthus emblica*

Family: Phyllanthaceae

Chemical constituents: Ascorbic acid, Tannins, Phytochemicals

Use: Amla is a fantastic cholesterol-lowering and detoxifying supplement. Its anti-inflammatory and free radical scavenging properties may aid in reestablishing the body's hormonal equilibrium.



Fig.Amla

### 8. Pumpkin seeds

Biological name: *Cucurbita pepo*

Family: Cucurbitaceae

Chemical constituents: Palmitic acid, linoic acid

Use: The beneficial omega-3 fatty acids found in pumpkin seeds can also help control the elevated insulin and cholesterol levels associated with PCOS. Additionally, they contain beta-sitosterol, which helps eliminate excess androgens and treat PCOS symptoms like weight gain, acne, and hirsutism [92].



Fig.Pumpkin seeds

### 9. Curcumin

Biological name: *Curcuma longa*

Family: Zingiberaceae

Chemical constituents: Tumerone, Zingiberone

Use: In female Wistar rats with PCOS produced by letrozole, curcumin shown positive effects. Its effects were similar to those of clomiphene citrate, the most popular medication for PCOS patients seeking to induce ovulation.



Fig.Curcumin

### 10. Tulsi

Biological name: *Ocimum sanctum*

Family: Lamiaceae

Chemical constituents: Linalol, eugenol, camphor, citral

Use: The absence of the ovulation process means that androgens are not used. Additionally, the liver produces a relatively small amount of SHBG protein. This explains why women struggle to conceive and have acne and excessive facial hair growth. Tulsi can reduce insulin levels and regulate androgens. It's a great antioxidant as well. On an empty stomach, chew at least ten leaves first thing in the morning. Regularly consume boiled tulsi water.



Fig.Tulsi

### 11. Ginger

Biological name: *Zingiber officinalis*

Family: Zingiberaceae

Chemical constituents: Phenolic acid, terpene compounds.

Use : Relieve nausea and vomiting and aid digestion.

### 12. Ginseng

Biological name: *Panax ginseng*

Family: Araliaceae

Chemical constituents: Ginseng saponin

Use: Estradiol Valerate (EV) was injected intramuscularly into female Sprague-Dawley rats weighing between 200 and 210 g to develop polycystic ovaries. These rats were then divided into three groups: EV control (n = 10), EV plus Ginseng Total Saponins (n = 10), and oil control (n = 10). Nerve Growth Factor (NGF) protein expression and ovarian morphology were noted. Rats with polycystic ovary syndrome showed increased expression of Nerve Growth Factor in both their brains and ovaries. The injection of ginseng whole saponins reduced the ovaries' expression of NGF [93].



Fig. 1.3: Ginseng

### 13. Black Cohosh Root

Biological name: *Cimicifuga racemosa*

Family: Ranunculaceae

Chemical constituents: Ferulic acid, caffeic acid

Use: Because it contains phytochemicals that can inhibit the secretion of luteinizing hormone, this plant has a powerful influence on the endocrine system. PMS, severe menstrual cramps, and hormone-related symptoms can all be effectively treated with black cohosh [94]

### 14. Gymnema

Biological name: *Gymnema sylvestre*

Family: Apocynaceae

Chemical constituents: Gymnemic acid, tartaric acid, calcium oxalate

Use: Gymnema is a traditional Ayurvedic herb that is used to decrease cholesterol, treat diabetes, promote weight loss, and lower blood sugar levels. Gymnema may have a trophorestorative effect on the pancreatic beta cells. The leaf is the part of the plant that is utilized as medication. Because of its ability to modulate insulin and the additional benefit of lowering the high triglycerides linked to PCOS, Gymnema is a good choice for PCOS [95–98].



Fig .Gymnema

### 15. Spearmint Tea

Biological name: *Menatha spicata*

Family: Labiatae

Chemical constituents: Limonene

Use: The investigation was conducted as a 30-day randomized controlled experiment in two centers in Turkey. For a month, 42 individuals were randomly assigned to drink spearmint tea twice



Fig. 1.2: Spearmint

daily, and their experiences were contrasted with a herbal tea placebo. Serum gonadotropin and androgen hormone levels were measured at 0, 15, and 30 days of the study; the degree of hirsutism was clinically assessed. Of the 42 patients, 41 finished the study. Over the course of 30 days, the spearmint tea group's levels of free and total testosterone as well as their level of hirsutism decreased. FSH and LH levels rose. The antiandrogen qualities of spearmint have been proven and validated [99].

## 10. Herbal drugs benefits in the treatment of Polycystic Ovary Syndrome (PCOS):

### • Hormonal Balance

1. Regulate menstrual cycles
2. Improve fertility
3. Reduce androgen levels
4. Balance estrogen and progesterone levels

### • Insulin Sensitivity

1. Improve glucose uptake
2. Reduce insulin resistance
3. Lower blood sugar levels

### • Anti-Androgenic Effects

1. Reduce hirsutism (excessive hair growth)
2. Improve acne
3. Reduce male pattern baldness

### • Antioxidant and Anti-Inflammatory Effects

1. Reduce oxidative stress
2. Improve inflammation markers

### • Weight Management

1. Aid in weight loss
2. Improve metabolic rate
3. Reduce body mass index (BMI)

### • Mood and Emotional Benefits

1. Reduce stress and anxiety
2. Improve mood swings
3. Enhance mental well-being

### • Other Benefits

1. Improve skin health
2. Reduce risk of cardiovascular disease
3. Enhance immune function [100-104].

## 11. Life Style Modification

### A) Exercise

The purpose of the study was to determine whether ovarian shape and sympathetic marker expression were affected by five weeks of voluntary exercise in the rat model of PCO induced by estradiol valerate (EV). In rats with EV-induced PCO, the impact of exercise was assessed on (i) ovarian morphology; (ii) nerve growth factor (NGF) mRNA and protein expression; and (iii) ovarian expressing cells for the NGF receptor (p75 neurotrophin receptor) and the  $\alpha 1a$ -,  $\alpha 1b$ -,  $\alpha 1d$ , and  $\beta 2$ -adrenergic receptors (ARs). In adult cycle rats, PCO was induced by a single intramuscular injection of EV, while controls received an injection of oil only. There were four groups of rats: (i) oil control; (ii) oil + exercise; (iii) PCO group (EV); and (iv) PCO exercise group (EV + exercise). In the cages where they were kept, the exercise and PCO exercise groups ran willingly for five weeks on computer-monitored wheels. According to the results, the PCO exercise

group's ovarian morphology was nearly normal, its NGF mRNA and protein concentrations were normal, its high numbers of NGF receptor-expressing cells in PCO ovaries were reduced by exercise, and its immunopositive cells of all the different AR subtypes—aside from the  $\alpha$ 1b- and  $\beta$ 2-AR—were all reduced after exercise, while the mRNA levels remained unchanged, suggesting transcriptional regulation. In summary, our findings suggest that regular exercise can help prevent and treat PCOS in humans by modulating ovarian sympathetic innervation [105].

## B) Dietary intake

Low glycemic index diet may help to manage insulin resistance, cardiovascular risk and irregular menstrual patterns in women with PCOS. One should avoid saturated fat intake. Lifestyle modification, including effective exercise regimens and dietary advice, should be the first line of treatment in women with polycystic ovary syndrome [106-107].

## Conclusion:

An ovarian cyst is a health condition becoming very common now a days. Nature has given us so many herbal remedies used for the treatment of PCOS. Synthetic medication have shown excellent management for the treatment of PCOS, but substantial adverse drug reactions on long term use. To enhance recovery rates and acceptance, patients are increasingly relying on herbal therapy as an alternative to synthetic medication for the control and treatment of PCOS. The herbal drugs are safe and provide therapeutic action without side effects on prolong and long term use. In today's era PCOS is emerging as a common problem in our society and herbal medication can play an important role in its management.

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