AYUSHDHARA
ISSN: 2393-9583 (P)/ 2393-9591 (O)
An International Journal of Research in AYUSH and Allied Systems

Review Article

POLYCYSTIC OVARIAN SYNDROME AND ENDOMETRIAL RECEPTIVITY CURRENT PERSPECTIVES AND SOLUTION THROUGH AYURVEDA: A REVIEW

Nidhi Bajpai[1]*, Anuradha Roy[2], Binay Sen[3]
*[1]Ph.D Scholars, [2]Associate Professor, Department of Prasuti Tantra, Faculty of Ayurveda, IMS BHU, Varanasi.
[3]Assistant professor, Department of Dravya Guna, Faculty of Ayurveda, BHU, Varanasi.

Article info
Article History:
Received: 21-07-2023
Revised: 05-08-2023
Accepted: 19-08-2023
KEYWORDS:
Endometrial receptivity, Poor obstetrical outcome, Polycystic ovarian syndrome, Ayurveda.

ABSTRACT

PCOS is a heterogeneous endocrine disorder which is characterised with oligomenorrhoea, anovulation, hirsutism and acne. Its prevalence increased exponentially from 2% to 18% in last two decades. On Later stages of its pathogenesis it manifests its metabolic involvement and causes various fertility issues and results into implantational failure which indicates endometrial defects. Conventional treatment includes combined oral contraceptives, insulin sensitizing drugs, anti-androgens and anti estrogenic agents etc. In alternative medicine various single herbs are mentioned which can reverse the pathology of PCOS and improve pregnancy outcome. So this review mainly rules out potential indicators for hampered endometrial receptivity and their solution through single herbs. **Material and Methods:** This is a literature review. Patient population is diagnosed cases of PCOS. This is a non interventional study. Data search was done from PubMed, Embase, and Cochrane Library. **Conclusion:** Patients with PCOS predisposed for endometrial receptivity disorder which can be cure with the use of single herbs via improved receptivity markers. Thus it helps to improve fertility and obstetrical outcome.

INTRODUCTION

The purpose of the study is to evaluate existing literature for possible association between polycystic ovarian syndrome (PCOS) and defective endometrial receptivity and their possible solutions through Ayurveda.

The design of the study is a literature review. The patients were women included in selected studies due to a diagnosis of PCOS associated with infertility and adverse pregnancy outcome. This study is non-interventional, with main outcome as to measure the association between endometrial factor and PCOS. A review of the literature relevant to it with its impact on overall female health was conducted. We conducted a search of published literature from various databases, such as PubMed, EMbase and Cochrane libraries. For this we searched data in two steps.

Access this article online	
Quick Response Code	
	https://doi.org/10.47070/ayushdhara.v10i4.1299

In first step the following MeSH terms in combination with polycystic ovarian syndrome: endometrial receptivity, sub-fertility, infertility, abortions, fertility agents and fertility therapy was searched. In second step following MeSH terms in combination with PCOS: Ayurvedic therapy, herbs, alternative solutions was searched.

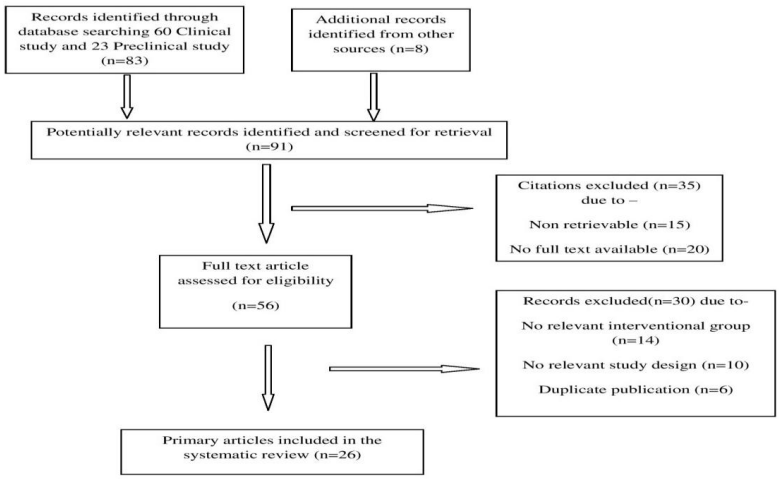
PCOS is a reproductive syndrome which also associated with other systematic dysfunctions. Its prevalence rate is approx 11% worldwide[1] and infertility found in approximately 40% of women with PCOS[2]. Altered endometrial function and ovulation defects both are important causes of PCOS associated infertility. However, change in endometrium remains unattended as a responsible factor of infertility as like ovulatory disorder[3]. It is observed that after restoration of ovulation pharmacologically conception rate is not as expected[4].

In PCOS patients recruitment of pre-ovulatory small follicles occurs. These follicles were not able to respond towards the normal physiological concentration of follicle stimulating hormone, and thus hinder the formation of dominant follicles[5]. Androgen can inhibit the growth and differentiation of

endometrial cells and the decidualization of the endometrium, thereby interfering with embryo implantation. Progesterone hormone concentration should dominate during "window of implantation", but patients with PCOS have lower level of progesterone due to chronic ovulatory defects, which ultimately alters the receptivity of the endometrium[6]. Focus on endometrial function may frame the basis of PCOS-related infertility or poor obstetrical outcomes[7]. The present study framed mainly for disorders of endometrial receptivity in patients with PCOS, where we planned to review the previous published literature to establish the altered endometrial receptivity markers in PCOS and their possible solution through Ayurveda in today's perspective.

PCOS is a multi-factorial disease. It involves pituitary dysfunction[8]. Enhanced ovarian androgen production[9], development of insulin resistance[10], obesity[11,12,13], birth weight and the adipose tissue expandability[14], leptin defects [15], oxidative stress [16], genetic [17,18,19,20], environmental [21], dietary [22], stress and other psychological factor[23].

Chart: Showing process of Review



OBSERVATIONS

S.No	Research	Design	Country	Inclusion	Sample size	Indicators used	Results
1.	Amooee S. et al.[24]	Retrospective cross-sectional study	Iran, 2020	PCOS patients	70	Endometrial histology, FSH, LH, PRL, TSH, testosterone, FBS, BMI and duration of infertility	Normal hysteroscopy findings but different histological findings.
2.	Wang et al.[25]	Clinical Study	China, 2022	PCOS patients	43	The mRNA expression levels of endometrial receptivity- related molecules were detected using reverse transcription-quantitative PCR.	Decreased mRNA expression levels of adiponectin receptor, ER, PR, IL-15, integrin avβ3. Increased mRNA expression levels of IL-6 and IL-8.
3.	Essam R et al. [26]	A prospective pilot case-control study	Egypt 2018	PCOD patients and fertile females	100	E.T. and pattern combined with Doppler examination of the uterine vessels for RI and PI. Evaluation of endometrial and sub-endometrial blood flow by 3DPD.	All 3DPD indices were significantly higher in fertile obese women. Uterine artery RI being higher in PCOD obese women than fertile obese women, while PI didn't show significant difference.

4.	Gao Q et.al[27]	Experimental study	China,2022	PCOS patients with HA	25 PCOS patients (13 with hyperandrogenism and 12 without hyperandrogenism) and 13 controls.	Gene expression profiling characteristics of PCOS with HA and NHA and identified immune-related factors that correlated with embryo implantation failure	DAPK2 was identified and validated as an independent decisive factor and critical biomarker associated with implantation failure, immune disorder and granulosa cell dysfunction implying poor implantation outcomes in HA PCOS.
5.	Kara M. et al.[28]	Controlled Clinical trial	Turkey, 2019	Diagnosed PCOS patients	53 patients of which 33 patients with PCOS.	Endometrial sampling in proliferative phase HOXA–10, HOXA–11, and LIF gene expressions were measured.	HOXA-10, HOXA-11, LIF mRNA expression levels in endometrial glandular epithelial cells were significantly lower in patients with PCOS.
6.	Ha LX et al.[29]	Controlled Clinical trial	China,2021	Diagnosed PCOS patients	162 (80 patients with PCOS and 82 patients with other gynaecological diseases)	TNF-α levels in the serum and uterine fluid were detected using enzyme-linked immune sorbent assay.	The BMI, AMH, LH, T, FINS, HOMA-IR,TG and LDL of patients with PCOS were higher. The TNF-α levels in the serum and uterine fluid of patients with PCOS were higher.
7.	D. Abdulkhalikova et al.[30]	Prospective case-crossover open-label study	Slovenia, 2022	PCOS patient with infertility	12	Endometrial sampling during implantation window for proteome determination.	Increased protein abundance was recorded for Legumain, Insulin-like growth factor-binding protein 7, Hepatocyte growth factor receptor, Keratin, type II cytoskeletal 7, and Cystatin-B, The B-lymphocyte antigen CD20 protein abundance decreased.
8.	Hu M. et al.[31]	Case control study	Swedan 2018	Diagnosed PCOS patients	25 PCOS (n = 14) without PCOS (n = 11)	PGR isoforms (PGRA and PGRB), estrogen receptor alpha (ERα), and markers of cell proliferation was determined by qRT-PCR, Western blot, immunohistochemistry and immunofluorescence assays.	PGRA mRNA and protein expression was higher in PCOS patients. PGRA/B and PGRB were localized in both epithelial and stromal cells, with notable changes in the nuclei of epithelial and

							stromal cells. A similar expression pattern of ERα, vimentin and Ki-67, in association with an increased PGR expression, was observed in PCOS patients.
9.	Younas K. et al. [32]	Cross over open label study	UK, 2019	Ovulatory and non ovulatory PCOS patients	116(65 PCOS and 51 fertile female	MAGEA11, and the genome-wide role of AR	MAGEA11 was present in epithelial and stromal compartments of fertile endometrium, with expression restricted to the cytoplasm and only low levels of MAGEA11 mRNA were detected in whole tissue extracts. In PCOS patients MAGEA11 levels were significantly higher.
10.	Mai M. et al. [33]	A prospective interventional study.	Egypt 2018	Diagnosed PCOS	38 obese and 21 lean	BMI, serum apelin, glucose, insulin, FSH, LH, T and HOMA-IR levels.	Significantly higher BMI and serum apelin were found in obese compared to lean PCOS.
11.	Paravati R et al. [34]	Prospective study	UK 2020	Control group- Parous women. Infertile group- women with PCOS	93 patients: 50 fertile. Infertile-43 with PCOS	Blood samples collected at mid proliferative phase- for routine fertility assessment of the hormonal panel and at secretary phase- progesterone, CD44, OPN and inflammatory cytokine levels.	LH was significantly higher and an FSH:LH ratio of <1 higher levels of androstenedione and DHEASwas detected in both PCOS groups. Abnormal CD44-OPN adhesion complex formation.

[Where FSH-follicle stimulating hormone, LH-Luteinizing hormone, PRL- Prolactin, TSH-Thyroid stimulating hormone, BMI- Body mass index, uNK-uterine natural killer cells, IR- insulin resistance, HA-hyperandrogenism, ER- oestrogen receptor, PR-progesterone receptor, IL- interleukins, NHA- Non-hyperandrogenism, NIR- non-insulin resistance, RI-Resistance index, PI-pulsatility index,3DPD-3 dimensional power doppler, DAPK2- Death associated protein kinase 2,HOXA- home box A cluster gene, AMH-anti Mullerian hormone, T- testosterone, FINS-fasting insulin, HOMA-IR-homeostasis model assessment insulin resistance, TG- triglyceride, LDL- low density lipoprotein, HOXA-11, LIF- leukaemia inhibitory factor, TNF- tumour necrosis factor, PGRA & PGRB-progesterone receptor A & B, DHEAS-dehydroepiandtrosterone sulphate.]

Solutions through Ayurveda

According to Ayurveda, *Artava* and *Ashaya dushti* may be considered for sub fertility or infertility. The pathogenesis of PCOS comes under the umbrella of entire etio-pathogenesis of both *Ashaya* and *Artava dushti* leading to subfertility. *Aartava-kshaya* is a disorder with involvement of obstruction in the *Apana Kshetra* with involvement of *Medovaha, Rasavaha, Raktavaha* and *Artava Vaha Srotas*. Therefore poly cystic ovarian syndrome can be understood as per concept of Ayurveda with the characteristics of *Dosha, Dhatu* and *Upadhatu.* So the treatment principles can be adopt which can clear the *srotorodha*, both at micro

and macro level (obstruction of channels), normalizes metabolic activity and even maintaining the *Rasa dhatu* which is prime factor of *Aartava kshaya*. In this review we collected evidences for single herbs action with its specific mechanism in patients of PCOS.

Evidences from Preclinical studies

S.No.	Name of the drug	Study design	Participants	Method	Form	Dose	Result
1.	*Cocos nucifera* [35]	Experimental	Female virgin wistar rats	Estrous cycle and blood sugar monitered once a week	Flower extract	100-200mg/kg of *cocos nucifera* flower extract	Recovered the estrous cycle; reduced TC, very low density cholesterol and TG levels; and increased HDL-C levels
2.	*Aloe barbadensis* miller [36]	Experimental	Letrozole induced PCOS rat model	Aloe vera gel for 2 months (10mg dry weight orally/60 days/daily), which was followed by induction of pregnancy and assayed for biosynthetic and metabolizing enzymes of steroidogenesis.	*Aloe vera* gel	*Aloe vera* gel for 2 months (10 mg dry weight orally/60 days/daily)	Altered ovarian-placental steroid status by modulating the expression of Steroidogenic acute regulatory (StAR), LHR, ARand Aromatase, also reduces post implantation loss during gestation period
3.	*Linum usitatissimum* L[37]	Experimental	Twenty four rats	Four groups including negative control, positive control, PCOS and treatment groups. Positive control group.	Hydroalcoholic extract of flaxseed	Flax seed extract 7 weeks after induction of PCOS for 30 days.	Increased antral follicles count and corpus luteum, Decreased number of cystic follicles, and reduced diameter of antral follicles
4.	*Zingiber officinale* [38]	Experimental study	63 adult female rats (170-200 gr) divided 9 groups.	7 experimental groups receiving estradiol valerate (PCOS inducing agent, intramuscular) alone and with 100mg/kg clomiphene or different doses of ginger extract.	Zinger extract	175 and 350 mg/kg) orally daily for 60 and 89 days	Lowered the levels of LH and estrogen, and increased the levels of FSH and progesterone in neonatal female SD rats
5.	Soy isoflavones [39]	Experimental	Sprague-Dawley rats	Physical parameter(Body wt., Uterus and ovary wt., Metabolic parameters (OGTT, Total cholesterol), Steroidal hormone profile, oxidative stress, Histopathology of ovary	Isoflavones	50 and 100mg/kg for 14 days	Improve symptoms as decreased body weight, testosterone and oxidative stress. Histological results reveal well developed antral follicles and normal granulosa cell layer in rat ovary

Evidences from Clinical Studies (Whole drug)

S.No	Name of Drug	Study design	Participants	Method	Dose	Results
1.	*Tribulus terrestris* [40]	RCT	Healthy women n = 8 early menstrual cycle (follicular phase)	concentration for FSH, LH Pre and post serum hormone testosterone and oestradiol at 8 am and 12 pm.	Intervention consisted of *Tribulus Terrestris* 250 mg per day over five days.	Showed significant increase in FSH, LH, oestradiol and no change in testosterone concentration.
2.	*Mentha spicata* [41]	RCT of 30 days	42	Ferriman-Galwey score and modified DQLI (At 0, 15 and 30 days of the study serum androgen hormone levels and gonadotrophins were checked.	Spearmint tea (5gm/250ml)twice a day for 1 month period	Free and total testosterone levels were significantly reduced. FSH:LH ratio increased. subjective assessments on degree of hirsutism scored by the modified DQLI were significantly reduced in the spearmint tea group
3.	*Glycerrhiza glabra* [42]	Single arm clinical trial	32	USG	7 grams powder per day	Improved ovulation rates in polycystic ovaries
4.	*Vitex agnus* castus[43]	A triple blind, RCT	70	Sr. Testosterone, Prolactin	Fruit extract 3 months	Normalize menstrual cycle duration in 68.6% the LD group and 60% of the extract participants. Decreased free testosterone, prolactin, mood changes and spotting
5.	*Cinnamon zeylanicum* [44]	RCT	84	FBS, HOMA-IR, Cholesterol	Bark powder prepared capsule 8 weeks	Decreased FBS, insulin, HOMA-IR, cholesterol, LDL and weight
6.	*Trigonella foenum-*graecum [45]	Open label, one-arm study	50 diagnosed with PCOS	Sonographic scan LH (IU/L) FSH (IU/L) LH/FSH ratio Waist circumference	Seed extract, Furocyst, 2 capsules of 500 mg each/day)	46%-reduction in cyst size, while 36% of subjects showed complete dissolution of cyst. 71% of subjects reported the return of regular menstrualcycle on completion of the treatment and 12% of subjects subsequently became pregnant
7.	*Linum usitatissimum* [46]	RCT	32	USG parameters, Hirsutism, Blood sugar	Flax seed powder 3 months	Decreased ovarian volume, number of antral follicle,regulates

						menstrual cyclicity and pregnancy
8.	*Foeniculum vulgare* [47]	RCT	61	Subjective parameters	Fennel seed infusion for 6 months	Decreased menstrual cycle interval and decreased dysmenorrhoea

DISCUSSION

The term ‘‘endometrial receptivity’’ refers to the ability of the uterine lining to accept and accommodate a nascent embryo, resulting in a successful pregnancy. As a highly dynamic tissue, the endometrium is periodically shed in response to the secretion of estrogens and progesterone. Due to underlying pathology as in pituitary dysfunction there is an increase in frequency and amplitude of GnRH pulse which results in elevated levels of LH. Enhanced ovarian androgen production, abnormal LH influence on the theca cell of the ovaries results in over production of androgens. Insulin insensitivity is intrinsic to PCOS. Obesity contributes significantly to both insulin resistance and hyperandrogenism. These are independent predictor of change from normoglycemia to impaired glucose tolerance and type 2 diabetes mellitus, contribute to a significant proportion of menstrual disorders. Birth weight and the adipose tissue expandability hypothesis imply that the degree of insulin resistance among normal weight women increased, these women are metabolically obese normal weight. The relationship of this with PCOS explained by birth weight and adipose tissue expandability hypothesis. Leptin is a hormone secreted by adipose tissue, serum level of these influenced by obesity, insulin resistance and the levels of sex steroids and insulin. It involved in regulation of body weight by decreasing appetite and increasing energy expenditure. Elevated levels of reactive oxygen species in follicular fluid and reduced antioxidant capacity are closely associated with reduced oocyte maturation and low embryo quality. Genetic factor is a complex oligogenic disorder in which a small number of key gene interact with environmental factors and manifests various phenotype. Existing treatment modalities to PCOS are limited due to the various clinical presentations in PCOS women, in some patient’s treatment failure occur, and in some may cause severe side effects. This drawback of current therapy drags interest towards alternative treatment. It is a natural way to treat the diseased condition without causing any serious side effects.

From the above cited studies histological assessment of PCOS patients found superior than hysteroscopy findings, mRNA expression study showed decreased levels of adiponectin receptor, ER, PR, IL 15, integrin avβ3, but increased mRNA expression levels of IL 6 and IL 8. In Doppler parameters PCOS patients have higher VI, FI and uterine artery RI**,**In Gene expression study HOXA 10, HOXA-11, LIF in endometrial glandular epithelial cells were significantly lower in patients with PCOS while Mage 11 were significantly higher, abnormal expression of DAPK2 in PCOS might cause abnormal recruitment of NK cells, impaired folliculogenesis, implantation failure and other poor pregnancy outcomes. ELISA showed higher TNF-α levels in the serum and uterine fluid. The body mass index, anti-Müllerian hormone, luteinizing hormone, testosterone, fasting insulin, homeostasis model assessment insulin resistance (HOMA-IR), triglycerides, and low density lipoprotein, androstenedione, free testosterone and dehydroepiandtrosterone sulphate of PCOS patients were higher. Proteomics study concluded that increased protein abundance for Legumain, insulin-like growth factor-binding protein 7, Hepatocyte growth factor receptor, Keratin, type II cytoskeletal 7, and Cystatin-B, while B-lymphocyte antigen CD20 was decreased. Immunohistochemistry and western blot analysis revealed PGRA mRNA and protein expression for ERα, vimentin and Ki-67 was higher in PCOS patients. In molecular analysis there was abnormal circulation of CD44-OPN adhesion complex and STAT 1. Also it is found that altered NF-κB pathways interactions modulate endometrial receptivity. On reviewing preclinical studies of single herbs *Narikela* (Cocos nucifera) mentioned as *Brinhan* (bulk enhancer), *Balya* (strength provider), *Bala mansa prad* (enhance strength and muscle mass), *Shukralam* (semen increasing), *Hridyam* (substance enhancing health and functioning of heart). In animal study it is found that it recovers the estrous cycle; reduces TC and very low density cholesterol. *Kumari* (Aloe vera) is *Raktashodhak* (blood purifier), *Shothahar* (substance alleviating inflammation), *Artavjanak* restoring menstrual flow), *Garbhasravkar* (promotes abortion), *Twakdoshahara* (removes skin ailments), and *Brinhan* (nourishing). It decreases testosterone levels and improves progesterone levels, modulates expression of Luteinizing hormone receptor (LHR), Androgen Receptor (AR) and Aromatase. Also reduces implantational loss which increases uterine receptivity and foetal growth. It also has potential role to sensitize the insulin receptor and reduce insulin level. So it can revert insulin resistant status by improved HOMA-IR

change. It has rich phytoesterols and phyto-phenols components which stimulates steroid metabolizing enzyme which helped in restoration of the ovarian structure-function to normalcy leading to improved fertility index. *Sunthi* (*Zingiber officinale*) mentioned as *Deepaniya* (appetizer), *Shoolprashman* (alleviates pain) it reduces cholesterol level which could decrease synthesis of steroid hormones such as estradiol, Thus leading to lower levels of LH and increased levels of FSH and progesterone. Soy isoflavones decreases body weight, testosterone and oxidative stress and increases antral follicle count. On reviewing clinical studies of single herb in PCOS clinical pathological condition of patients. *Gokshura* (*Tribulus terrestris*) mentioned under *Shothahar* (substance alleviating inflammation). Classically indicated for sexual function, anti diabetes, anti-inflammatory, antitumor and antioxidant effects. It can be proved on the basis of steroidal saponins and flavonoids which acts as anti-aging and anti-inflammatory, so by these actions it can improve insulin resistance, reduces intracellular promoter of inflammation–transcription factor nuclear factor kappa B (NF-kB) and improves LH and increases estradiol level. *Podina* (*Mentha spicata*) has properties of *Rechana* (therapeutic purgation), *Vatakapha nashak* (alleviates *Vata* and *Kapha dosha*), *Rajah pradah* (enhance menstrual flow). It acts on weight reduction, have hypoglycaemic, hypolipidemic and anti androgenic action. It contains flavonoids, phenol, glycoside, terpenoids and steroidal compounds which may act over beta cells for insulin production and utilization and also reduces absorption of glucose. It reduces atrietic follicles and ovarian cyst size and work on lowering testosterone level and hirsutism. *Yashtimadhu* (*Glycyrrhiza glabra*) mentioned under *Varnya* (promotes complexion), *Shonitsthapan* (hemostatic) *mahakashaya*. It is leguminous and its roots been used since long time for chest and lung disease, kidney and heart disease, fluid retention, low blood pressure, allergies, hyperglycemias, leucorrhoea and skin diseases. Its main ingredients are triterpene, saponins and flavonides which shows biological activities such as antioxidant, dermatological activity. Glabridin inhibited melanogenesis by inhibition of ROS and inhibit tyrosine so it can cure hyper pigmentation in PCOS, isoliquiritigenin reduces inflammatory response of macrophages improves ovulation rate. *Nirgundi* (*Vitex agnus* castus) has *Keshya* (beneficial for hairs), *Kushtha hara* (alleviates skin problems), *Katipradeshastha vata nashak* (regulates *Vata dosha* in pelvic area) properties. It has essential oil, alkaloids, and other phytoconstituents that have prominent role over pituitary gland and lowered prolactin level, improve menstrual cycle, improve low progesterone level, hot flashes, and reduces testosterone and LH level. It helps in PCOS by modulating kisspeptin gene expression and normalizes menstrual cycle. *Dalchini* (*Cinnamon zeylanicum*) mentioned in *Eladi gana* has *varnya, Kaphavatnashak, Rajah sravi* (stimulate blood flow), *Garbhashaya sankochak* (uterine contractor) properties. It decreases FBS, insulin, HOMA-IR, cholesterol, LDL and weight. *Mishreya* (*Foeniculum vulgare*) has *Vrashya* (aphrodisiac), *Vatapitta hara, Balya* properties. It decreases menstrual cycle interval and dysmenorrhoea. It has been conventionally used for anovular infertility. It has strong anti-inflammatory, estrogenic and antioxidant property. It has role over LH: FSH ratio, DHEAS level, ovarian follicles. *Methi* (*Trigonella foenum*-graecum) is *Deepaniya* (appetizer), *Vatanashak, Vajikaran* (aphrodisiac) properties. It helps to reduce ovarian volume, improve LH and FSH ratio, decreases blood sugar levels and triglycerides. *Atasi* (*Linum usitattisimum*) has *Vatanashak* (alleviates *Vata dosha*), *Ushna-veerya* (hot potency), *Balya, Twak doshanashak* properties. It contains lignans which decreases androgen levels and also helps in normalising lipid level, effective in the growth and development of follicles, and corpus luteum and reduces the cyst follicles. Flaxseed supplementation can help women to control androgen levels and decreases hirsutism.

CONCLUSION

PCOS is the most prevailing female endocrine disorder and prime cause of infertility. In India its prevalence is 3.7-22.5%. Studies showed importance of histopathology and molecular markers to diagnose endometrial defects. These can be proved as potent endometrial receptivity markers. Ayurvedic single herbs acts on various factors of PCOS via altering these endometrial receptivity markers and by removing obstruction in channels (*Alasi, Gokshura, Nirgundi*) and improving *Rasa dhatu* (*Kumari, Dalchini, Methi, Sunthi, Narikela*). So, it can be concluded that as per the pathology of PCOS and underlying clinical condition plants mentioned above can be used to cure the problem and also data suggests beneficial effects over various underlying morbid pathology which can predispose PCOS patients for various fertility issues.

REFERENCES

1. Bharali MD, Rajendran R, Goswami J, Singhal K, Rajendran V. Prevalence of Polycystic ovarian syndrome in india: A Systematic review and meta-analysis. Cureus.2022 Dec 9; 14(12): e32351.
2. Patel S. Polycystic ovary syndrome (PCOS), an inflammatory, systemic, lifestyle endocrinopathy. J Steroid Biochem Mol Biol. 2018; 182:27–36. Epub 2018/04/22.
3. Jiang NX, Li XL. The Disorders of Endometrial Receptivity in PCOS and Its Mechanisms. Reprod sci. 2021 May 27

4. Silvana R. Ferreira and Alicia B. Motta, Uterine Function: From Normal to Polycystic Ovarian Syndrome Alterations Volume 25, Issue 15, 2018
5. Xue Z, Li J, Feng J, Han H, Zhao J, Zhang J, Han Y, Wu X, Zhang Y. Research Progress on the Mechanism Between Polycystic Ovary Syndrome and Abnormal Endometrium. Front Physiol. 2021 Dec 17; 12: 78
6. Zhang H., Song X., Han Y., Xue F., Yang Z. (2007). Analysis of endometrial pathological status in patients with polycystic ovary syndrome. Chin. J. Obstet. Gynecol. 42, 493–494.
7. Nidhi Bajpai, Anuradha Roy, Binay Sen. Subfertility in Polycystic ovarian syndrome and role of Ayurveda: A case report. J of Ayurveda and Hol Med (JAHM). 2022; 10(6): 71-79
8. Madnani N, Khan K, Chauhan P, Parmar G. Polycystic ovarion syndrome. Indian J Dermatol Venereol Leprol, 2013; 79: 310-21.
9. Ibid
10. Ibid
11. Isikoglu M, Berkkanoglu M, Cemal H, Ozgur K, Polycystic ovarian syndrome: what is the role of obesity?, UK, Anshan publisher, 2006; 157-163.
12. Dahlgren E, Janson PO, Johansson S et. al, Polycystic ovarian syndrome and risk for myocardial infarction. Evaluated from a risk factor model based on a prospective population study of women. Acta Obstet Gynecol Scand, 1992; 71: ruo] 599-604
13. Mastorakos G, Koliopoulos C, Creatsag G. Androgen and lipid profiles in adolescents with polycystic ovarian syndrome who were treated with two forms of combined oral contraceptives. Fertility and Sterility. 2002; 77(5): 919-927.
14. Bernier, Danielle. Polycystic ovarian syndrome: pathogenesis, health consequences and treatment of PCOS in relation to insulin resistance, 2012. Honors Theses and Capstones; 3: 15-17.
15. Gautam N. Allahbadia, Rubina Merchant; Polycystic ovary syndrome and impact on health, Middle East Fertility Society Journal, 2011; 16: 19-37.
16. V. De Leo, M.C. Musacchio, V. Cappelli et. al. Genetic, hormonal and metabolic aspects of PCOS: an update, Reproductive Biology and Endocrinology, 2016; 14: 38-45.
17. Frank S, Gharani N, McCarthy M. Genetic abnormalities in polycystic ovarian syndrome. Ann Endocrinol Paris, 1999; 60: 131-133.
18. Legro RS, Strauss JF, Molecular progress in infertility: polycystic ovarian syndrome. Fertile Sterile 2002; 78: 569-576.
19. Arora S, Allahabadia GN. Familial association in women with polycystic ovarian syndrome, UK, Anshan publisher; 2006: 85-90
20. Kaushal R, Parchure N, Bano G et.al. Insulin resistance and endothelial dysfunction in the brothers of Indian subcontinent Asian women with polycystic ovarian syndrome. Clin. Endocrinol (Oxf), 2004; 60: 322.
21. Escobar-Morreale HF, Luque-Ramirez et.al. The molecular genetic basis of functional hyperandrogenism and the polycystic ovarian syndrome. Endocr Rev 2005; 26: 251-282.
22. De Melo AS, Dias SV et.al. Pathogenesis of polycystic ovarian syndrome: multifactorial assessment from the foetal stage to menopause. Reproduction 2015; 150: 11-24
23. Woodruff TJ, Janseen S, Guillette et.al. Environmental impacts on reproductive health and fertility. New York: Cambridge University Press; 2010
24. Amooee S, Akbarzadeh-Jahromi M, Motavas M, Zarei F. "Comparing endometrial hysteroscopic and histological findings of infertile women with polycystic ovary syndrome and unexplained infertility: A cross-sectional study," Int J Reprod Bio Med 2020; 18: 33–40. https://doi.org/10.18502/ijrm.v18i1.6195
25. Wang, C., Wen, Y., Mai, Q."Impact of metabolic disorders on endometrial receptivity in patients with polycystic ovary syndrome". Experimental and Therapeutic Medicine 23, no. 3 (2022): 221. https://doi.org/10.3892/etm.2022.11145
26. Essam R. Othman, Karim S. Abdullah, Ahmed M. Abbas, Mostafa Hussein, Elwany Elsnosy, Ihab H. El-Nashar, Evaluation of endometrial and subendometrial vascularity in obese women with polycystic ovarian disease, Middle East Fertility Society Journal, Volume 23, Issue 4, 2018, 324-330, ISSN 1110-5690, https://doi.org/10.1016/j.mefs.2018.04.006.
27. Gao Q, Ma C, Meng S, Wang G, Xing Q, Xu Y, He X, Wang T and Cao Y. Exploration of molecular features of PCOS with different androgen levels and immune-related prognostic biomarkers associated with implantation failure. Front. Endocrinol. 2022; 13: 946504. doi: 10.3389/fendo.2022.946504.
28. Kara M, Ozcan SS, Aran T, Kara O, Yilmaz N. Evaluation of endometrial receptivity by measuring HOXA-10, HOXA-11, and leukemia inhibitory factor expression in patients with polycystic ovary syndrome. Gynecol Minim Invasive Ther, 2019; 8: 118-22.
29. Ha LX, Li WX, Du YD, Yuan YY, Qu XX. Tumor Necrosis Factor Alpha Level in the Uterine Fluid of Patients with Polycystic Ovary Syndrome and Its Correlation with Clinical Parameters. J Inflamm Res. 2022 Oct 29; 15: 6015-6020. doi: 10.2147/JIR.S382808. PMID: 36339827; PMCID: PMC9628701.
30. Abdulkhalikova D., Sustarsic A., Vrtacnik Bokal Eda, Jancar N., Jensterle M., Burnik Papler T. The Lifestyle Modifications and Endometrial Proteome Changes of Women With Polycystic Ovary Syndrome and Obesity. Frontiers in Endocrinology. 2022; 13

https://www.frontiersin.org/articles/10.3389/fendo.2022.

31. Hu M, Li J, Zhang Y, Li X, Brännström M, Shao LR, Billig H. Endometrial progesterone receptor isoforms in women with polycystic ovary syndrome. Am J Transl Res. 2018 Aug 15; 10(8): 2696-2705. PMID: 30210706; PMCID: PMC6129510.
32. Younas K, Quintela M, Thomas S, Garcia-Parra J, Blake L, Whiteland H, Bunkheila A, Francis LW, Margarit L, Gonzalez D, Conlan RS. Delayed endometrial decidualisation in polycystic ovary syndrome; the role of AR-MAGEA11. J Mol Med (Berl). 2019; 97(9): 1315-1327. doi: 10.1007/s00109-019-01809-6.
33. Mai M. Hasan, Azza A. Abd El Hameed, Serum adipokine (apelin) in lean and obese polycystic ovary syndrome patients before and after metformin treatment, Middle East Fertility Society Journal, 2018; 23(4), 315-318, 1110-5690, https://doi.org/10.1016/j.mefs.2018.04.003.
34. Paravati R, De Mello N, Onyido EK, Francis LW, Brüsehafer K, Younas K, Spencer-Harty S, Conlan RS, Gonzalez D, Margarit L. Differential regulation of osteopontin and CD44 correlates with infertility status in PCOS patients. J Mol Med (Berl). 2020 Dec; 98(12): 1713-1725. doi: 10.1007/s00109-020-01985-w.
35. V. Soumya, Y. Indira Muzib, P. Venkatesh, K. Hariprasath. GC-MS analysis of Cocus nucifera flower extract and its effects on heterogeneous symptoms of polycystic ovarian disease in female Wistar rats [J]. Chinese Journal of Natural Medicines, 2014, 12(9): 677-684.
36. Radha MH and Laxmipriya NP. Role of Aloe Barbadensis Mill. as a Possible Pre-Conceptive Herb for the Management of Polycystic Ovarian Syndrome: A Rodent Model Study. Austin J Reprod Med Infertil. 2016; 3(2): 1040.
37. Jelodar G, Masoomi S, Rahmanifar F. Hydroalcoholic extract of flaxseed improves polycystic ovary syndrome in a rat model. Iran J Basic Med Sci. 2018 Jun; 21(6): 645-650.
38. Atashpour S, KargarJahromi H, KargarJahromi Z, Maleknasab M. Comparison of the effects of Ginger extract with clomiphene citrate on sex hormones in rats with polycystic ovarian syndrome. Int J Reprod Biomed. 2017 Sep; 15(9): 561-568.
39. Rajan RK, M SS, Balaji B. Soy isoflavones exerts beneficial effect on letrozole induced rat polycystic ovarian syndrome (PCOS) model through anti-androgenic mechanism. Pharm Biol. 2017; 55(1): 242-251.
40. Milanov S, Maleeva A, Tashkov M. Tribestan effect on the concentration of some hormones in the serum of healthy subjects. Sofia, Bulgaria: Company documentation, Chemical Pharmaceutical Research Institute; 1981.
41. Grant P. Spearmint herbal tea has significant anti-androgen effects in polycystic ovarian syndrome. A randomized controlled trial. Phytother Res. 2010; 24(2): 186–188.
42. Lee JC, Pak SC, Lee SH, Lim SC, Bai YH, Jin CS, Kim JS, Na CS, Bae CS, Oh KS. The effect of herbal medicine on nerve growth factor in estradiol valerate-induced polycystic ovaries in rats. *Am J Chin Med.* 2003; 31(06): 885–895.
43. Shahnazi M, Khalili AF et al. The effect of combined low dose oral contraceptives and Vitex agnus on the improvement of clinical and paraclinical parameters of polycystic ovarian syndrome.: A triple blind, randomized, controlled clinical trial. Iran Red Crescent Med J. 2016; 18(12).
44. Borzoei A, Rafraf M, Asghari-Jafrabadi M. Cinnamon improves metabolic factors without detectable effects on adiponectin in women with polycystic ovarian syndrome. Asia Pac J Clin Nutr. 2018; 27(3): 556-63.
45. Swaroop A, Jaipuriar AS, Gupta SK, Bagchi M, Kumar P, Preuss HG, Bagchi D. Efficacy of a Novel Fenugreek Seed Extract (Trigonella foenum-graecum, Furocyst) in Polycystic Ovary Syndrome (PCOS). Int J Med Sci. 2015 Oct 3; 12(10): 825-31.
46. Farzana F et. Al. Effect of flax seed supplementation in polycystic ovarian syndrome. J Res Med Sci. 2015; 31(1): 113-9.
47. Mokaberinejad R, Rampishesh Z, Aliasl J, Akhtari E, The comparison of fennel infusion plus dry cupping versus metformin in management of oligomenorrhoea in patients with polycystic ovarian syndrome: a randomised clinical trial. J Obstet Gynaecol, 2019: 1-7

Cite this article as:
Nidhi Bajpai, Anuradha Roy, Binay Sen. Polycystic Ovarian Syndrome and Endometrial Receptivity Current Perspectives and Solution Through Ayurveda. AYUSHDHARA, 2023;10(4):26-35.
https://doi.org/10.47070/ayushdhara.v10i4.1299
Source of support: Nil, Conflict of interest: None Declared

***Address for correspondence**
Dr. Nidhi Bajpai
Ph.D Scholars,
Department of Prasuti Tantra,
Faculty of Ayurveda, IMS BHU,
Varanasi.
Email:
drnidhibajpai90@gmail.com