

Review Article

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Diagnosis and treatment of polycystic ovarian syndrome: an update

Abstract

Polycystic ovarian syndrome (PCOS) and polycystic ovarian disease (PCOD) affect women's reproductive health. Specifically, PCOS involves elevated androgens, leading to abnormal oocyte growth, ovulation, and ovary fluid accumulation. Likewise, PCOD entails hormonal imbalances that lead to the formation of follicular cysts and the retention of immature eggs. Such symptoms include menstrual irregularities, obesity, hirsutism, depression, infertility, and excessive facial hair growth. The illnesses affect over 116 million women globally. Although the exact reasons are unknown for certain, elevated ratios or LH/ FSH and GnRH rates could contribute to this. Treatment strategies include medication, surgery, changes in lifestyle, and exploration of alternative therapies for getting hormone levels under control and handling symptoms. Diagnosis is made by using a medical history, physical examination, blood tests, and ultrasound. They are advised to maintain a healthy diet and minimize exposure to unhealthy meals.

Keywords: polycystic ovary syndrome/polycystic ovary disease, hyperandrogenism, insulin resistance, hirsutism, infertility, ultrasound, laparoscopy, laparotomy, diet

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Introduction

WHO estimates that more than 116 million women, representing about 3.4 percent of the female populace, suffer from polycystic ovarian syndrome globally (PCOS). This endocrine condition leads to excessive secretion of androgens, which interrupts egg development and ovulation in the ovaries. Therefore, they form ovarian cysts.¹ The reported incidence of PCOS in the population ranges from 6 to 10%, depending on the criteria used to characterise it.² Likewise, PCOD affects approximately 22.5% of women; it is a condition that results from the hormonal abnormality causing the immature eggs to collect in the ovary, leading to the formation of cysts.

The symptoms of PCOS and PCOD are almost identical; these include an irregular menstruation cycle, obesity, the development of facial hair, depression, infertility, and hirsutism. Exactly what triggers these diseases remains obscure, but it is thought that hormonal aberrations, such as elevated levels of luteinizing hormone (LH) to follicle-stimulating hormone (FSH) ratio and increased frequency of the gonadotropin-releasing hormone (GnRH).³ PCOS has nonreproductive morphidities, like obesity, metabolic syndrome, hyperinsulinemia, insulin resistance, hepatic steatosis, and dyslipidemia that make women with this disease to be at higher risk of cardiovascular diseases and type 2 diabetes. Nevertheless, the pathophysiology of PCOS is still unknown, making mechanism-based therapies unreachable despite the disease's high incidence and substantial negative effects on health.⁴

The treatment of PCOS and PCOD is geared towards managing symptoms and restoring hormonal balance. Birth control pills, hormone treatments, and insulin-sensitizing medicine are some of the medications that can be used.³ In cases that are more serious, surgical interventions like laparoscopy could be investigated. Also, people should engage themselves in exercise frequently and maintain healthy diets to control these diseases.

Symptoms

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Hirsutism is a condition where there is excessive hair growth on the face and body. Hair thinning, darkness of skin where skin creases that

is neck, arm pits, and groin. Menstrual irregularity, heavy bleeding when periods occur, severe obesity, ovarian cysts, and acne on the forehead and under the chin. Some others are depression, headache, infertility, fatigue, low sex drive, mood swings, male pattern baldness, migraine, and severe pelvic pain.⁵

Aetiology

PCOS is an oligogenic disorder whose clinical, biochemical, and heterogeneous phenotypes are determined by the interaction of several genetic and environmental aspects. PCOS is characterised by an unknown genetic actiology and often runs in families.⁶ Many environmental factors of PCOS are related to poor dietary practices, non-physical activity levels, infectious agents, and toxins.7 Lifestyle changes like weight reduction can also reverse some procreative and metabolic complications associated with PCOS.8 As androgen levels increase, it affects the LH and FSH ratio, which causes follicular arrest and dysplasia. Metabolic syndrome emerges from these factors causing hyperinsulinemia, hyperandrogenism, oxidative stress, and irregular periodicity, leading to an increase in metabolic syndrome. PCOS is a term that was coined after women with numerous ovarian cysts were noted in an ultrasound examination. Primitive follicles are developed, but because of disrupted ovarian function, they cease at a very early stage.9

Pathomechanism of the PCOS

Hyperandrogenism is experienced by most women with PCOS, with rates ranging from 60 to 80 percent. The side effects of high androgen production include hirsutism and hyperandrogenism. On the contrary, hyperandrogenism is the most conspicuous feature found in PCOS condition, which is a key player in the involved hormone disruptance that governs the pathogenesis of PCOS. One prevalent sign of hyperandrogenism is higher concentration of free testosterone levels in bloodstream.¹⁰

Primary ovarian pathophysiology

These interactive influences on follicular growth commonly leads to the development of just one follicle that undergoes successive terminal maturation and ovulation. The number of preantral ovarian

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follicles is much lesser during birth; it is around 2 to 3 million when it was about 6-7 million at the middle of the gestational period. The ovarian reserve and fertility can only be sustained by regulating the rate of recruitment of new primordial follicles into the growing pool. Still, these follicles are gradually shed from it.11 There is a dynamic balance between dormant and active follicles. This study also revealed that overall follicle arrest in PCOS patients is due to the fluctuation in the level of anti-Müllerian hormone (AMH), follicle-stimulating hormone (FSH), and androgens.¹² When the level of luteinizing hormone (LH) is high, theca cells synthesize androgens. However, low FSH levels accompanied by inability of androgens to be transported to oestradiol means that no dominant follicle can be selected and therefore, prolonged anovulation occurs.¹³ This balance is regulated by the secretion of AMH hormone from granulosa cells whereby AMH inhibits the growth of primordial follicular to primary follicle. Consequently, PCOS is characterized by an increase in the size of the small follicles, which in turn stagnates and acquires a polycystic appearance. There are theories that suggest that the follicles in PCOS ovary are different from those in normal ovaries.14

Insulin Resistance (IR)/Hyperinsulinemia

Hyperinsulinemia and insulin resistance (IR) have been reported in women with PCOS irrespective of their androgen levels or BMI in the overweight range.¹⁵ Women in this group are also at a high risk for acquiring poor glucose tolerance and type 2 diabetes.¹⁶ Notably, females suffering with PCOS encounter tissue-selective IR. Insulin sensitivity triggers steroidogenesis in the ovary and the adrenal gland. On the other hand, the tissues such as the liver, skeletal muscle, and adipose tissues become less sensitive to the impact of insulin on the metabolic regulation. Thus, although steroid-producing tissues remain sensitive to insulin, some tissues in female PCOS patients acquire insulin resistance.17 Hyperinsulinemia and insulin resistance may also be caused by other reasons for instance; elevation of testosterone levels experienced during puberty. Previous investigations have focused on the association between hypoandrogenicity and IR, primarily because of the connection between rare autoimmune diseases affecting insulin receptors and hypoandrogenic signs.18 Elevated levels of diacylglycerol (DAG) and ceramides in the liver and muscle interfere with the insulin signaling pathway. Intracellular ceramides can also have a detrimental impact on insulin signaling by inhibiting the translocation of protein kinase B (Akt), a vital regulator of insulin sensitivity to the plasma membrane.¹⁹ However, it is imperative to emphasize that the dysfunction of insulin regulation in the central nervous system is connected to obesity and suboptimal development of ovarian follicles, suggesting that there can be other connections between obesity, PCOS and hyperinsulinemia in animals.²⁰

Neuroendocrine alterations

Gonadotropin secretion alterations in PCOS: Gonadotropins LH and FSH, which regulate ovulation, follicle development, and steroidogenesis in the ovary, are discovered and secreted peculiarly in PCOS females, although they are not necessary for diagnosis. Thus, it is conceivable that modified secretion of gonadotropins might have impact on hyperandrogenism and ovulatory dysfunction, which are the core signs of PCOS. Moreover, it has been reported that females with PCOS have raised luteinizing hormone levels in blood stream, enhanced LH pulse frequency or amplitude, high LH/FSH ratios, and relatively low FSH levels.^{21,22} However, some females have PCOS with hyperandrogenism, particularly those who are obese, additionally possess non-elevated baseline or provoked LH levels, demonstrating the variety of symptoms (and aetiology) associated with the disease. While the dissociation between gonadotropin-releasing hormone

(GnRH) and leptin (LH) had been observed within numerous models, this might potentially clarify why several obese females along with PCOS produce reduced levels of LH compared to other individuals, even though LH is considered to be the indicator of GnRH intervals.²³

Other metabolic and endocrine factors of GnRH secretion in PCOS

According to current study, AMH performs a hitherto unknown role in the positive control of GnRH-secreting nerve cells. It has been demonstrated that central administration of AMH has a dosedependent impact of stimulating the pulsatile production of LH in mice female. The anti-Müllerian hormone receptor type 2 (AMHR2) receptors in nerve cells releasing GnRH are activated by the GnRHdependent action for AMH. In this context, excessive LH production could be influenced by the uncontrolled levels of anti-Müllerian hormone (AMH) in PCOS. Despite the possibility that AMH plays a key role in PCOS, AMH-induced neurosecretion of GnRH has only been shown in control animals-neither PCOS models nor patients.²⁴ These neuroendocrine changes can also be caused by elevated insulin levels and insulin resistance, notwithstanding hyperandrogenism and potentially other ovarian factors are primary aspects for enhanced GnRH/LH secretion.25 Insulin infusion enhanced the frequency of LH pulses in control females, in accordance with the secretory sequences in females with PCOS. Furthermore, it has been shown that slender woman with PCOS have higher baseline LH levels, which raises the LH/FSH proportion. A further study that included females with PCOS discovered that insulin therapy had no impact on the pulsatility of LH.^{25,26}

Diagnosis

Blood tests

Hormonal blood tests

- I. The most reliable diagnostic tests for hyperandrogenism are blood tests for testosterone and the free androgen index (FAI).
- II. Blood tests to identify male sex hormone levels presence: for the presence of Sex hormone-binding globulin (SHBG), Dehydroepiandrosterone sulphate (DHEAS) and Androstenedione.
- III. Venipuncture to check for the levels of other reproductive hormones that could be impacting menstrual cycle like Estradioloestrogen (the main female sex hormone), Follicle-stimulating hormone (FSH), Luteinizing hormone (LH)
- IV. Symptoms of PCOS can be analyzed by measuring the levels of the following hormones, Thyroid-stimulating hormone (TSH), and Lactotropin
- V. Hormones related to suprarenal function (glands found above the kidney) Example: 17-hydroxyprogesterone.²⁷

Sonography

Sonography entails imaging of the uterus, ovaries, and pelvis. The photo indicates whether cysts exist or not, and show if the ovary has been enlarged. Endometrial lining sampling is a useful test that could indicate risk of womb cancer for people who experience very irregular menstrual cycles. However, if mensural cycles are irregular, there are indices or measurement showing high amounts of androgen and if the patient is below 20 years, ultrasound scan in not necessary. When there is abnormal degree of androgens or atypical symptoms such as severe alopecia, hirsutism, and menstrual irregularities, it requires assistance of ultrasonography. **Transvaginal ultrasound:** However, a transvaginal ultrasound is limited to sex-active females; otherwise, it is an abdominal scan. Transvaginal ultrasound is an absolutely pain-free, non-radiation test. The tool engaged is a probe which is inserted in the vagina with an ultrasonic sensor attached on top of it. The image quality is better than that usually produced by abdominal ultrasonography.²⁸

Abdominal ultrasound: It helps to display the female gonads externally from behind the abdominal wall (Figure 1).



Figure I Comparison of ultrasound between normal ovary (a) and PCOD/ PCOS ovary $(b)^{29}$

Alternative tests

If the female is diagnosed with PCOS, it is essential to assess the risks for heart problems and diabetes, as PCOS leads to insensitivity towards insulin and obesity, some of the tests are, Blood pressure, Glucose metabolism/tolerance blood test, and Lipid panel test.

Females diagnosed with PCOS will have to go for annual blood pressure checks, cholesterol tests, and diabetes tests after one to three years. You will need these tests more often if you have risk factors such as a heredity of diabetes and past anomalous lipid profile tests.²⁸

Treatment

Anovulation condition

Clomiphene: Ovulation in PCOS/PCOD is induced by Clomiphene citrate; initially, 50 mg/day oral tablets for 5 days are administered. The mechanism of clomiphene citrate is not known. When ovulation occurs but there is no sign of pregnancy symptoms, 50 mg/day is given for five (5) consecutive days. But when there is an absence of ovulation, the dose is increased to 100mg for five consecutive days, not less than thirty days after a prior course of treatment. Possible side effects comprise ovarian enlargement, ovarian hyperstimulation syndrome, bloating, and discomfort.³⁰

Aromatase inhibitor

Letrozole: Non-steroidal competitive inhibitor of aromatase, an enzyme involved in the conversion of androgens into estrogens. Ovulation is induced. Dizziness, bone pain, increased sweating, and headaches are the side effects.³¹

Antidiabetic drugs

Metformin: For the treatment of insulin resistance and restoring irregular menses in PCOS/PCOD. Thus, it enhances the uptake and utilisation of glucose in a woman suffering from PCOS/PCOD. Metformin works indirectly by decreasing the level of insulin by curbing the activity of the CYP17 cytochrome, which results in reduced production of androgens. The ovulation rate and pregnancy rate were shown to increase when taken along with clomiphene citrate. These side effects may include nausea, stomach diarrhoea, abdominal pain, and headaches.³²

Oral contraceptives: These pills can be categorised into progesterone-only pills or combined pills that contain both oestrogen and progesterone. This is why they are the frontline therapy for women to avoid unwanted pregnancies. OCs treat PCOD/PCOS by regulating the menstruation period. This minimises hirsutism, acne, and androgen levels.³³

Natural treatment for PCOS/PCOD

There are many ayurvedic and homoeopathic medications for the treatment of PCOS/PCOD.

Ayurvedic medication

Aspagarus racemosus: Its general name is shatavari. It is predominantly used to stimulate the normal growth and maturation of the ovarian follicles. It helps to regularize menstruation and restore strength in women's reproductive systems. It helps to regulate very high insulin levels caused by PCOS as it has phytoestrogens, or natural plant-based oestrogen levels.

Tinospora cordifolia: Its common name is guduchi, which is an Ayurvedic herb and has potent anti-inflammatory properties that help in the management of PCOS problems. Chronic inflammation in body tissues leads to an imbalance in insulin levels and the formation of cysts in the ovaries of women. It contains an ayurvedic herb used to rejuvenate the body and reduce insulin resistance.

Foeniculum vulgare: Its accepted name is fennel seeds. The phytoestrogens in the fennel seeds lessen inflammation and regulate insulin resistance, thereby functioning like a supplement.

Triphala: One of the effective natural remedies for patients suffering from PCOD symptoms is triphala. It is an ancient Ayurvedic medicine that contains vitamin C, which is a strong natural antioxidant with the ability to reduce inflammation by eliminating free radicals. Therefore, this treatment is an ideal detoxification method and should be used before other Ayurvedic medicine for better results.

Aloe Vera-Kumari (Aloe barbadensis): Another very effective ayurvedic medicine for the treatment of PCOS is Aloe Vera, which helps regulate menses and stimulates normal menstruation. It also normalizes hormonal imbalances.³⁴

Homoeopathic medication

Homoeopathy is very helpful for PCOS/PCOD treatment, definitely better than any other alternative medicine. In that respect, homoeopathic medicines provide a better remedy, as their primary aim lies in addressing the cause of the disease rather than just alleviating symptoms. They provide energy supplements, which aid in the healing and rebalancing of the life force and energy of the entire personality. Some of the medications that have proven to be effective in the treatment of PCOS/POCD.³⁵

- a) Pulsatilla is taken for PCOS with very scanty and delayed menstruation.
- b) Sepia is used to treat PCOS with severe pains
- c) Graphite is used in treating PCOD with constipation.
- d) Apis Mellifica is used for treating PCOS patients suffering from pricking pain.

Surgical method

Surgical extraction of big and continuous ovarian cysts is one of the very widely used approach. If one has worries about possible

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cancer in the cyst, surgery is usually suggested. These suregeries are generally performed under general anaesthesia.

There are two types of surgical methods to remove cysts:

Laparoscopy. Laparoscopy can remove most of the cysts. It is a keyhole surgery; this involves making tiny cuts near your navel as well as blowing up the pelvic cavity to enable the surgeon to get at your appendages. The doctor uses a specialised instrument called a laparoscope, which is a small tube-shaped microscope that contains a light at one tip for viewing the interior of an individual's abdominal cavity. He takes out the cyst using his surgical knife and goes through your tiny skin cuts. The dissolvable stitches are used to close the cuts after the cyst has been removed. Laparoscopy is preferred due to its lower pain and shortened recovery period. People may go back home immediately after undergoing this procedure. **Laparotomy:** Laparotomy may become necessary if your cyst happens to be bigger than ordinary measurements or has characteristics that suggest cancerous traits. It involves making a big incision during the operation where the abdominal cavity is opened up, hence exposing the cyst to offer the doctor more direct access. They can remove the whole cyst and ovary and take it to a lab to determine if it is malignant. The incision will then be closed by stitches or staples.³⁶

Other ways to cure PCOS /PCOD

Healthy lifestyle, regular exercise (walking for 40 minutes, cycling, jogging, etc.). Yoga is one of the best ways to treat reproductive diseases. Some of the asanas are Setu Bandhasana (Bridge Pose), Chakravakasana (Cat-Cow Pose), Malasana (Garland pose) Surya Namaskar Janusirsana (head-to-knee pose) along with healthy diet.

Food to eat and avoid for PCOS/PCOD patients

Table I PCOS/PCOD diet37

Foods to Eat	Foods to Avoid
Eat green, leafy vegetables like Spinach, collards, broccoli, cabbage, brussels sprouts, etc.	Avoid sugary foods like cakes, chocolates, drinks, sweets, etc.
Eat healthy fats (cow ghee).	
Essential fatty acids like tuna, salmon, nuts, and seeds like pumpkin seeds, chia seeds, flax	Avoid white flour in breads, cereals, bagels, and muffins.
seeds, etc.	
Fat protein-rich foods like eggs sovahean pork pulses totu lentils and seafood	Avoid unhealthy fats like trans-fat, saturated fats, and
Lat protein-nen loods inte e663, 307 abean, porte, puises, told, ientiis, and searood.	hydrogenated fats.
Eat fruits like cherries, plums, apricots, bananas, pomegranates, papayas, kiwis,	Avoid dairy products like cheese and butter.
strawberries, oranges, etc.	Avoid processed red meat like Sausages and hot dogs.
Vegetables like carrot, beetroot, tomatoes, eggplant, red bell pepper, mushrooms, and	
sweet potato	Avoid packed toods like junk tood (chips, hoodles, etc.).

Conclusion

The exact cause of PCOD/PCOS remains unclear, but several risk factors, such as obesity, inflammation, stress, inheritance patterns, and physical inactivity, could increase the susceptibility to developing this syndrome. Finally, after giving advice about some lifestyle changes and additional recommendations, symptomatic treatment is prescribed using multiple drugs that may include antidiabetics, antiandrogens, contraceptives, ayurveda and homeopathy medications.

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Conflict of interest

The authors declare that there is no conflict of interest.

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References

- Bharathi RV, Swetha S, Neerajaa J, et al. An epidemiological survey: effect of predisposing factors for PCOS in Indian urban and rural population. *Middle East Fertility Society Journal*. 2017;22(4):313–316.
- Waghmare SV, Shanoo A. Polycystic ovary syndrome: a literature review with a focus on diagnosis, pathophysiology, and management. *Cureus*. 2023;15(10):e47408.
- Bednarska S, Siejka A. The pathogenesis and treatment of polycystic ovary syndrome: What's new?. Adv Clin Exp Med. 2017;26(2):359–367.

- Shorakae S, Boyle J, Teede H. Polycystic ovary syndrome: A common hormonal condition with major metabolic sequelae that physicians should know about. *Intern Med J.* 2014;44(8):720–726.
- 5. MedicalNewsToday.
- Xita N, Georgiou I, Tsatsoulis A. The genetic basis of polycystic ovary syndrome. *Eur J Endocrinol*. 2002;147(6):717–725.
- Diamanti-Kandarakis E, Kandarakis H, Legro RS. The role of genes and environment in the etiology of PCOS. *Endocrine*. 2006;30(1):19–26.
- Shannon M, and Yusharn Wang. Polycystic ovary syndrome: a common but often unrecognized condition. J Midwifery Womens Health. 2012;57(3):221–230.
- Dumesic DA, Oberfield SE, Stener-Victorin E, et al. Scientific statement on the diagnostic criteria, epidemiology, pathophysiology, and molecular genetics of polycystic ovary syndrome. *Endocr Rev.* 2015;36(5):487–525.
- Ibáñez L, Oberfield SE, Witchel S, et al. An international consortium update: pathophysiology, diagnosis, and treatment of polycystic ovarian syndrome in adolescence. *Horm Res Paediatr*. 2017;88(6):371–395.
- Hsueh AJW, Kawamura K, Cheng Y, et al. Intraovarian control of early folliculogenesis. *Endocr Rev.* 2015;36(1):1–24.
- Franks S, Stark J, Hardy K. Follicle dynamics and anovulation in polycystic ovary syndrome. *Hum Reprod Update*. 2008;14(4):367–378.
- Gervásio CG, Bernuci MP, Silva-de-Sá MF, et al. The role of androgen hormones in early follicular development. *ISRN Obstet Gynecol.* 2014;2014:818010.
- Webber LJ, Stubbs S, Stark J, et al. Formation and early development of follicles in the polycystic ovary. *Lancet.* 2003;362(9389):1017–1021.

- Dunaif A, Segal KR, Shelley DR, et al. Evidence for distinctive and intrinsic defects in insulin action in polycystic ovary syndrome. *Diabetes*. 1992;41(10):1257–1266.
- Diamanti-Kandarakis E, Dunaif A. Insulin resistance and the polycystic ovary syndrome revisited: an update on mechanisms and implications. *Endocr Rev.* 2012;33(6):981–1030.
- Geffner ME, Golde DW. Selective insulin action on skin, ovary, and heart in insulin-resistant states. *Diabetes Care*. 1988;11(6):500–505.
- Moller DE, Flier JS. Insulin resistance—mechanisms, syndromes, and implications. N Engl J Med. 1991;325(13):938–948.
- Badin PM, Langin D, Moro C. Dynamics of skeletal muscle lipid pools. *Trends Endocrinol Metab.* 2013;24(12):607–615.
- Bruning JC, Gautam D, Burks DJ, et al. Role of brain insulin receptor in control of body weight and reproduction. *Science*. 2000;289(5487):2122– 2125.
- Azziz R. Polycystic ovary syndrome. Obstet Gynecol. 2018;132(2):321– 336.
- Taylor AE, McCourt B, Martin KA, et al. Determinants of abnormal gonadotropin secretion in clinically defined women with polycystic ovary syndrome. J Clin Endocrinol Metab. 1997;82(7):2248–2256.
- Moenter SM. Leap of faith: does serum luteinizing hormone always accurately reflect central reproductive neuroendocrine activity?. *Neuroendocrinology*. 2015;102(4):256–266.
- Cimino I, Casoni F, Liu X, et al. Novel role for anti-Müllerian hormone in the regulation of GnRH neuron excitability and hormone secretion. *Nat Commun.* 2016;7(1):1–12.

- Chavez JA, Summers SA. A ceramide-centric view of insulin resistance. *Cell Metab.* 2012;15(5):585–594.
- Moret M, Stettler R, Rodieux F, et al. Insulin modulation of luteinizing hormone secretion in normal female volunteers and lean polycystic ovary syndrome patients. *Neuroendocrinology*. 2009;89(2):131–139.
- 27. Verywell health. Blood tests for PCOS. 2024.
- 28. Jean Hailes for Women's Health.
- 29. MAUREEN MOOMJY. PCOS.
- CLOMID (clomiphene), prescribing information. Bridgewater, N.J.: Sanofi-Aventis U.S.; 2006.
- 31. Propecia® Drug Information. 2012.
- Bulsara J, Patel P, Soni A, et al. A review: brief insight into polycystic ovarian syndrome. *Endocrine and Metabolic Science*. 2021;3:100085.
- Ndefo UA, Eaton A, Green MR. Polycystic ovary syndrome: a review of treatment options with a focus on pharmacological approaches. *PT*. 2013;38(6):336–355.
- Lybrate. PCOD How Ayurveda medicines & treatment is beneficial for you?. 2023.
- 35. Amrita Homeopathy. PCOS/PCOD.
- 36. NHS. Ovarian cyst. 2023.
- 37. Care Hospital. PCOD diet chart: foods to eat and avoid. 2023.