

# POLYCYSTIC OVARY SYNDROME

<sup>1</sup>K.Malleswari\*,<sup>2</sup>D.Rama Brahma Reddy,<sup>3</sup>J. Rohith, <sup>3</sup>K. Ravindra, <sup>3</sup>K. Jamal Basha.

<sup>1</sup>Associate Professor, <sup>2</sup>Principal & professor, <sup>3</sup>Student, <sup>4</sup>Student, <sup>5</sup>Student.  
Department Of Pharmaceutics.

Nalanda Institute Of Pharmaceutical Scinces, Sathenapalli, Guntur, Andhra Pradesh, India.

**Abstract:** The polycystic ovary syndrome (PCOS) is the most common cause of anovulatory infertility and a notable proportion of women of reproductive age are affected. It may constitute a risk factor for cancer development. Different factors could result in different manifestations and many of these are related to predispositions. It is essential to establish criteria to achieve an exact diagnosis of PCOS, especially among adolescent patients because of the overlap between features of PCO syndrome and physiological findings in puberty. Day by day the technology of ultrasonography is improving and accuracy is increasing, but remains dependent on the specific equipment available. Some factors are inter-related in determining PCOS prognosis. Serum AMH is synthesized by small antral follicles, which are precisely those seen on ultrasound and could help us to diagnose PCOS but there are many aspects that still require elucidation. In this mini- review we have attempted to identify some of these correlations.

**Keywords:** Polycystic ovary syndrome, anti-Millerand hormone, diagnosis, treatment.

## I. INTRODUCTION:

*Polycystic ovary syndrome*, or *PCOS*, is the most common endocrine disorder in women of reproductive age. The syndrome is named after the characteristic cysts which may form on the ovaries, though it is important to note that this is a sign and not the underlying cause of the disorder.

Women with PCOS may experience irregular menstrual periods, heavy periods, excess hair, acne, pelvic pain, difficulty getting pregnant, and patches of thick, darker, velvety skin.<sup>[3]</sup> The primary characteristics of this syndrome include hyperandrogenism, an ovulation, insulin resistance, and neuroendocrine disruption. A review of the international evidence found that the prevalence of PCOS could be as high as 26% among some populations, though ranges between 4% and 18% are reported for general populations. Despite its high prevalence, the exact cause of PCOS remains uncertain and there is no known cure.

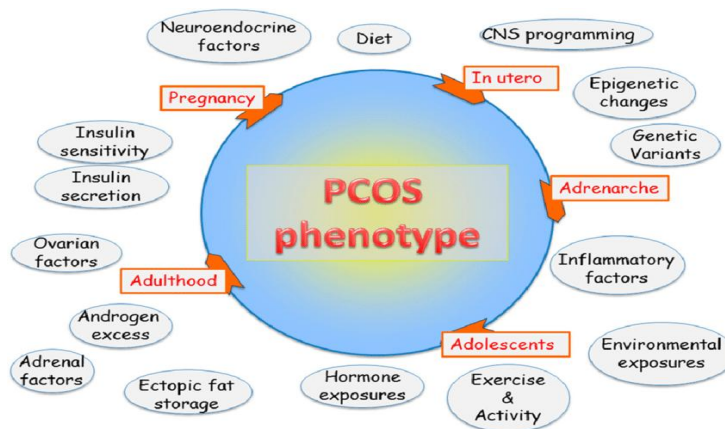


Fig:1 Polycystic ovary syndrome

**Cyst** is a closed sac, having a distinct envelope and division compared with the nearby tissue. Hence, it is a cluster of cells that have grouped together to form a sac (like the manner in which water molecules group together to form a bubble); however, the distinguishing aspect of a cyst is that the cells forming the "shell" of such a sac are distinctly abnormal (in both appearance and behaviour) when compared with all surrounding cells for that given location. A cyst may contain air, fluids, or semi-solid material. A collection of pus is called an abscess, not a cyst. Once formed, a cyst may resolve on its own. When a cyst fails to resolve, it may need to be removed surgically, but that would depend upon its type and location<sup>[6]</sup>

### Types of polycystic ovary syndrome:

- 1) Insulin-Resistant PCOS:** This is the classic type of PCOS and by far the most common. High insulin and leptin impede ovulation and stimulate the ovaries to make testosterone. Insulin resistance is caused by sugar, smoking, trans fat, and environmental toxins.
- 2) Pill-Induced PCOS or Post-Pill PCOS:** Hormonal birth control suppresses ovulation. For most women, it's a temporary effect, and ovulation will usually resume fairly soon after the Pill is stopped. But for some women, ovulation-suppression can persist for months or even years. During that time, it is not unusual to be given the diagnosis of PCOS. Some experts deny the existence of Pill-induced PCOS, but it is very real. It is the second most common type of PCOS.
- 3) Inflammatory PCOS:** Inflammation or chronic immune activation results from by stress, environmental toxins, intestinal permeability and inflammatory foods like gluten or A1 casein. Inflammation is a problem for PCOS because it impedes ovulation, disrupts hormone receptors, and stimulates adrenal androgens such DHEA and androstenedione.

**4) Hidden-Cause PCOS:** This is the 'simpler-than-you-think' type of PCOS. There is one simple thing that is blocking ovulation. Once that single thing is addressed, this type of PCOS resolves very quickly, usually within 3-4 months.

### I. ETIOLOGY:

PCOS can be described as an oligogenic disorder in which the interaction of a number of genetic and environmental factors determine the heterogeneous, clinical, and biochemical phenotype. Although the genetic etiology of PCOS remains unknown, a family history of PCOS is relatively common; however, familial links to PCOS are unclear. A lack of phenotypic information prevents a formal segregation analysis. Nonetheless, the current literature suggests that the clustering of PCOS in families resembles an autosomal dominant pattern.

Environmental factors implicated in PCOS (e.g., obesity) can be exacerbated by poor dietary choices and physical inactivity; infectious agents and toxins may also play a role. The reproductive and metabolic features of PCOS are sometimes reversible with lifestyle modifications such as weight loss and exercise.<sup>[13-14]</sup>

**What Causes Infertility in Women with Polycystic Ovarian Syndrome?**

Women with PCOS might not ovulate every month. The reason behind this is the hormonal imbalance. The tiny follicles that develop every month in the ovaries need a number of hormones to grow, develop, mature and rupture. In women with PCOS the ovaries does not produce some of the hormones in sufficient amount. These women also have problem in the hormone levels of Follicle Stimulating Hormone (FSH) and Luteinizing Hormone (LH) and the abnormal ratio of these two hormones also pose difficulty for the follicles to grow. The hormonal sufficiency and imbalance creates a lot of disturbance leading to under-maturation of the follicle which stays in the ovaries in the form of a cyst. These cysts in turn do not produce enough progesterone and cause irregularities in the menstrual cycle. The androgen produced in women with PCOS due to problems in insulin regulation also poses a problem in ovulation. The cycle continues leading to no ovulation and delayed menstrual cycle which is the chief reason for infertility for women with PCOS. Polycystic Ovarian Syndrome Affecting Pregnancy.

### II. EPIDIMIOLOGY:

Polycystic ovarian syndrome (PCOS) is one of the most common endocrine disorders of reproductive-age women, with a prevalence of 4-12%. Up to 10% of women are diagnosed with PCOS during gynecologic visits. In some European studies, the prevalence of PCOS has been reported to be 6.5-8%.

A great deal of ethnic variability in hirsutism is observed. For example, Asian (East and Southeast Asia) women have less hirsutism than white women given the same serum androgen values. In a study that assessed hirsutism in southern Chinese women, investigators found a prevalence of 10.5%. In hirsute women, there was a significant increase in the incidence of acne, menstrual irregularities, polycystic ovaries, and acanthosis nigricans.

PCOS affects premenopausal women, and the age of onset is most often perimenarchal (before bone age reaches 16 years). However, clinical recognition of the syndrome may be delayed by failure of the patient to become concerned by irregular menses, hirsutism, or other symptoms or by the overlap of PCOS findings with normal physiologic maturation during the 2 years after menarche. In lean women with a genetic predisposition to PCOS, the syndrome may be unmasked when they subsequently gain weight.<sup>[2]</sup>

#### ***Pioglitazone for treating polycystic ovary syndrome in non-obese women of reproductive age with different clinical presentations***

Twenty-eight women of reproductive age with PCOS were recruited; 20 women finished the study. The women were divided into three groups according to clinical presentations: group A (n = 4) had chronic oligo- or anovulation with polycystic ovaries; group B (n = 5) had chronic oligo- or anovulation with hyperandrogenism; and group C (n = 11) had chronic oligo- or anovulation, hyperandrogenism and polycystic ovaries. Pioglitazone (15 mg/day) was given for 6 months and the therapeutic effects were evaluated. Menstrual cycle regularity and hormone levels (plasma luteinizing hormone (LH), follicle-stimulating hormone, total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol, glucose, insulin, C-peptide, free testosterone, homeostatic model assessment (HOMA)) were evaluated during and after pioglitazone treatment.<sup>[2]</sup>

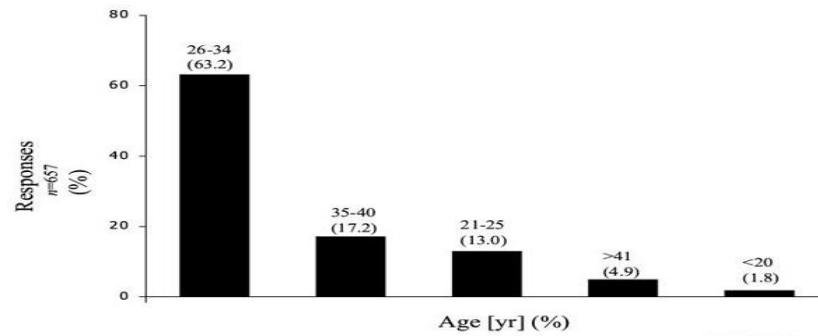
#### ***Thiazolidinediones for treatment of polycystic ovary syndrome***

Troglitazone has been evaluated in numerous clinical trials of women with PCOS. These trials provided a body of evidence supporting the efficacy of troglitazone for management of PCOS complications, such as insulin resistance, hyperandrogenism, and anovulation. Due to safety concerns, however, troglitazone is no longer marketed in the United States. Clinical data are emerging regarding the utility of newer, safer thiazolidinediones, such as pioglitazone and rosiglitazone, for this patient population. The available literature provides evidence that these newer agents improve insulin sensitivity, glycemic control, hormone responsiveness, menstrual regularity, and ovulation rates. Pioglitazone and rosiglitazone have been well tolerated in clinical studies and have an improved safety profile in terms of liver toxicity. Pioglitazone and rosiglitazone should be considered a second-line treatment alternative to metformin for management of women with PCOS who are resistant to insulin or who are obese.

#### ***An overview of polycystic ovary syndrome in aging women***

The manifestations of polycystic ovary syndrome (PCOS), a ubiquitous reproductive disorder, may vary significantly depending on the severity of a number of endocrine and metabolic changes. Although no diagnostic criteria are presently available for PCOS for perimenopausal and menopausal women, the condition can still be suspected in case of a previous diagnosis of the condition, a chronic history of irregular menstrual cycles and hyperandrogenism, and/or polycystic ovarian morphology during the reproductive period. PCOS is associated with long-term health risks, including obesity, diabetes, hypertension, dyslipidemia, metabolic syndrome and cardiovascular risk factors during reproductive age, especially in patients possessing classic phenotypes. The aim of this review was to outline the available data about the impact of PCOS on long-term health risks after reproductive age in patients with PCOS. Previously, it was assumed that women with PCOS would be more prone to develop cardiometabolic diseases after reproductive age but current data suggest that in accordance with the healing in the phenotypic characteristics of PCOS, no deterioration appears to occur in cardiometabolic health in these patients. While there is substantial evidence for a greater prevalence of abnormal subclinical atherosclerotic markers among younger patients with PCOS, data for older women are insufficient. However, there is also support for an increased risk of endometrial cancer in PCOS patients. Extensive prospective cohort studies in which healthy

controls as well as patients with defining PCOS phenotypes are observed and monitored from the early reproductive period into the late postmenopausal period should now be performed in order to clarify morbidities and mortality in aging women with PCOS.



Graph:1 Age distribution of polycystic ovary

### ***Polycystic Ovarian Syndrome as a Cause of Infertility***

Although there are many reasons posing a problem for conception, polycystic ovarian syndrome ranks the highest among all of them. Among the female factors of infertility, 75% of them have PCOS as the reason for their anovulatory infertility.

#### **What Causes Infertility in Women with Polycystic Ovarian Syndrome?**

Women with PCOS might not ovulate every month. The reason behind this is the hormonal imbalance. The tiny follicles that develop every month in the ovaries need a number of hormones to grow, develop, mature and rupture. In women with PCOS the ovaries does not produce some of the hormones in sufficient amount. These women also have problem in the hormone levels of Follicle Stimulating Hormone (FSH) and Luteinizing Hormone (LH) and the abnormal ratio of these two hormones also pose difficulty for the follicles to grow. The hormonal sufficiency and imbalance creates a lot of disturbance leading to under-maturation of the follicle which stays in the ovaries in the form of a cyst. These cysts in turn do not produce enough progesterone and cause irregularities in the menstrual cycle. The androgen produced in women with PCOS due to problems in insulin regulation also poses a problem in ovulation. The cycle continues leading to no ovulation and delayed menstrual cycle which is the chief reason for infertility for women with PCOS.<sup>[5]</sup>

### ***Polycystic Ovarian Syndrome Affecting Pregnancy***

Women who had polycystic ovarian syndrome are often at higher risk for various complications during pregnancy. A woman with PCOS must be educated by the physician about these risks. Higher rates of miscarriage, pregnancy induced high blood pressure, gestational diabetes, and premature deliveries are the common effects of PCOS on a woman who has achieved pregnancy. Moreover, a child born to a woman with PCOS are found to spend longer time in NICU and are at higher risk of dying before, during or just after birth and these chances are even higher in multiple-birth babies.

### **III. Pathogenesis:**

Polycystic ovaries develop when the ovaries are stimulated to produce excessive amounts of androgenic hormones, in particular testosterone, by either one or a combination of the following (almost certainly combined with genetic susceptibility). The release of excessive luteinizing hormone (LH) by the anterior pituitary gland. Through high levels of insulin in the blood (hyperinsulinaemia) in women whose ovaries are sensitive to this stimulus. The syndrome acquired its most widely used name due to the common sign on ultrasound examination of multiple (poly) ovarian cysts. These "cysts" are in fact immature ovarian follicles. The follicles have developed from primordial follicles, but this development has stopped ("arrested") at an early stage, due to the disturbed ovarian function. The follicles may be oriented along the ovarian periphery, appearing as a 'string of pearls' on ultrasound examination.

Women with PCOS experience an increased frequency of hypothalamic GnRH pulses, which in turn results in an increase in the LH/FSH ratio.

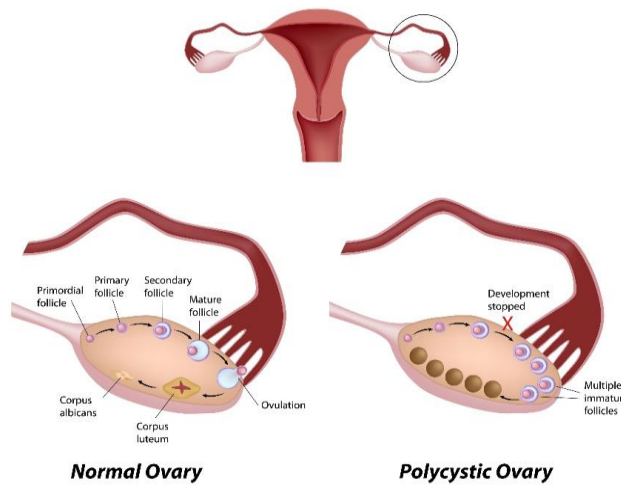
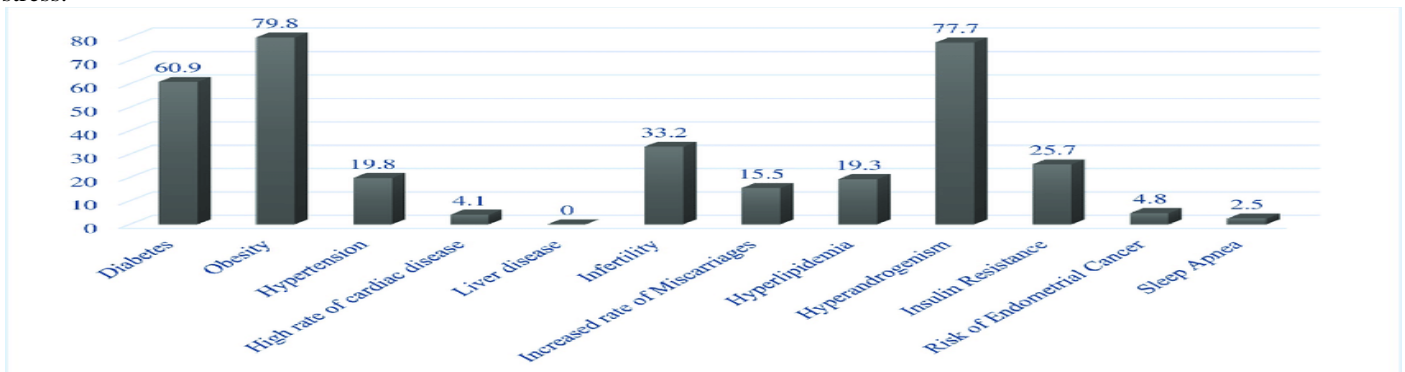


Figure 2: Normal and Polycystic ovary differentiation

A majority of women with PCOS have insulin resistance and/or are obese. Their elevated insulin levels contribute to or cause the abnormalities seen in the hypothalamic-pituitary-ovarian axis that lead to PCOS. Hyperinsulinemia increases GnRH pulse frequency; LH over FSH dominance; increased ovarian androgen production; decreased follicular maturation; and decreased SHBG binding. Furthermore, excessive insulin, acting through its cognate receptor in the presence of component CAMP signalling, up regulates 17 $\alpha$ -hydroxylase activity via PI3K, 17 $\alpha$ -hydroxylase activity being responsible for synthesising androgen precursors. The combined effects of hyper insulinemia contribute to an increased risk of PCOS. Insulin resistance is a common finding among women with a normal weight as well as among overweight women.

Adipose tissue possesses aromatase, an enzyme that converts androstenedione to estrone and testosterone to estradiol. The excess of adipose tissue in obese women creates the paradox of having both excess androgens (which are responsible for hirsutism and virilization) and excess estrogens (which inhibit FSH via negative feedback).

PCOS may be associated with chronic inflammation,<sup>1</sup> with several investigators correlating inflammatory mediators with anovulation and other PCOS symptoms. Similarly, there seems to be a relation between PCOS and an increased level of oxidative stress.<sup>[2-3]</sup>



Graph: 2 percentage of complication among patient with polycystic ovary syndrome related other diseases

**Metabolic consequences of obesity and insulin resistance in polycystic ovary syndrome: diagnostic and methodological challenges:**

Women with polycystic ovary syndrome (PCOS) have a considerable risk of metabolic dysfunction. This review aims to present contemporary knowledge on obesity, insulin resistance and PCOS with emphasis on the diagnostic and methodological challenges encountered in research and clinical practice. Variable diagnostic criteria for PCOS and associated phenotypes are frequently published. Targeted searches were conducted to identify all available data concerning the association of obesity and insulin resistance with PCOS up to September 2016. Articles were considered if they were peer reviewed, in English and included women with PCOS. Obesity is more prevalent in women with PCOS, but studies rarely reported accurate assessments of adiposity, nor split the study population by PCOS phenotypes

**Metabolic dysfunction in polycystic ovary syndrome: Pathogenic role of androgen excess and potential therapeutic strategies:**

Polycystic ovary syndrome (PCOS) is the most common endocrinopathy among reproductive age women. Although its cardinal manifestations include hyperandrogenism, oligo/anovulation, and/or polycystic ovarian morphology, PCOS women often display also notable metabolic comorbidities. An array of pathogenic mechanisms have been implicated in the etiology of this heterogeneous endocrine disorder; hyperandrogenism at various developmental periods is proposed as a major driver of the metabolic and reproductive perturbations associated with PCOS. However, the current understanding of the pathophysiology of PCOS-associated metabolic disease is incomplete, and therapeutic strategies used to manage this syndrome's metabolic complications remain limited. Androgen excess plays a prominent role in the development of metabolic disturbances associated with PCOS, with a discernible impact on key peripheral metabolic tissues, including the adipose, liver, pancreas, and muscle, and very prominently the brain, contributing to the constellation of metabolic complications of PCOS, from obesity to insulin resistance.

However, the current understanding of the pathogenic roles of hyperandrogenism in metabolic dysfunction of PCOS and the underlying mechanisms remain largely incomplete. In addition, the development of more efficient, even personalized therapeutic strategies for the metabolic management of PCOS patients persists as an unmet need that will certainly benefit from a better comprehension of the molecular basis of this heterogeneous syndrome.<sup>[7]</sup>

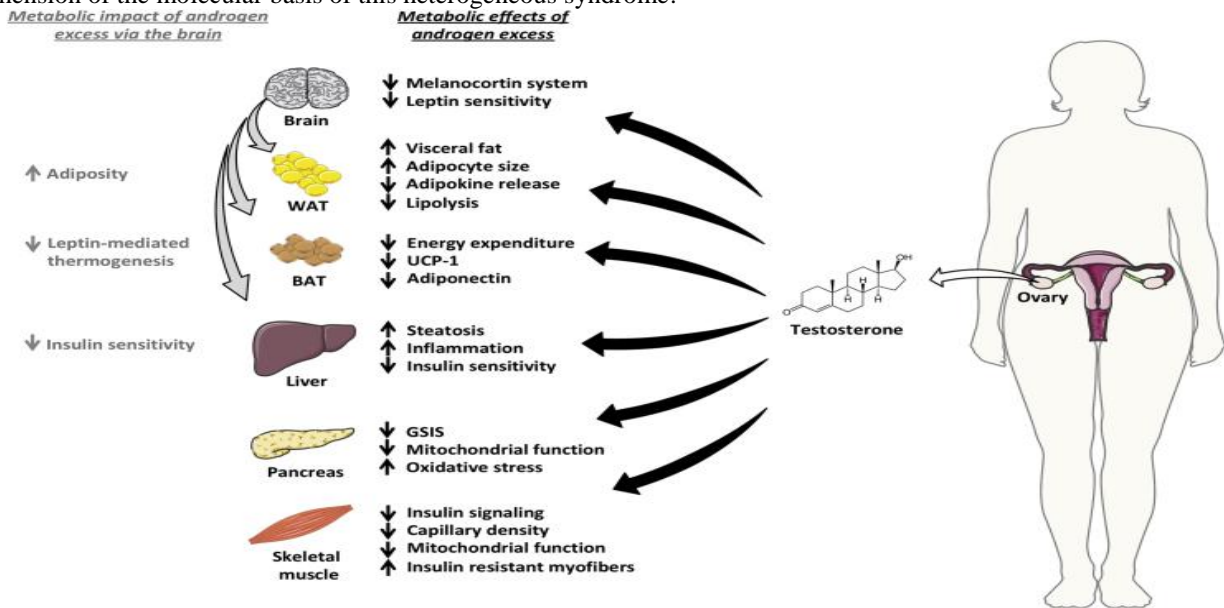


Fig No: 3 Metabolic effects of androgen in females.

#### Exploration of the classification of polycystic ovarian syndrome:

A cross-sectional study of 192 women with PCOS (14 - 38 years of age) was performed. The patients were divided into 3 groups of A, B and C according to the revised 2003 consensus on diagnostic criteria and also divided into 2 groups according to body mass index (BMI): group A (n = 110), long term anovulation, clinical and biochemical evidence of high androgen level, ovary enlargement with its size larger than 10 ml or number of small follicles of 2 - 9 mm  $\geq$  12 under ultrasound with exclusion of other diseases caused by high androgen; group B (n = 46), long term anovulation, clinical and biochemical evidence of high androgen level; group C (n = 36), long term anovulation, ovary enlargement with its size larger than 10 ml or number of small follicles of 2 - 9 mm  $\geq$  12 under ultrasound with exclusion of other disease caused by high androgen; obesity PCOS group (OB-PCOS, n = 70), BMI  $\geq$  25 (kg/m<sup>2</sup>); no obesity PCOS group (NOB-PCOS, n = 122), BMI < 25 (kg/m<sup>2</sup>). One hundred and four women with bilateral tubal block factor caused infertility served as control group. Anthropometric measurements, Ferriman Gallwey hirsutism scoring, presence of acne and acanthosis nigricans were noted. Hormonal profile was assessed by measuring follicle-stimulating hormone (FSH), luteinizing hormone (LH), free testosterone (FT), prolactin (PRL), sex hormone binding globulin (SHBG). The metabolic profile was investigated by measurements of oral glucose tolerance test (OGTT), serum lipid levels, including total cholesterol (Chol), triglycerides (TG), high-density lipoprotein (HDL), and low-density lipoprotein (LDL). Hyperinsulinemia was estimated by measurement of fasting insulin (FINS) and insulin area under the curve (IAUC). The extent of insulin resistance (IR) and hyperandrogenism was estimated by homeostasis model assessment (HOMA) and free androgen index (FAI) respectively.<sup>[8]</sup>

#### IV. SIGNS AND SYMPTOMS:

Signs and symptoms of PCOS include irregular or no menstrual periods, heavy periods, excess body and facial hair, acne, pelvic pain, difficulty getting pregnant, and patches of thick, darker, velvety skin. This metabolic, endocrine and reproductive disorder is not universally defined, but the most common symptoms are irregular or absent periods, ovarian cysts, enlarged ovaries, excess androgen, weight gain and hirsutism. Associated conditions include type 2 diabetes, obesity, obstructive sleep apnea, heart disease, mood disorders, and endometrial cancer. This disease is related to the number of follicles per ovary each month growing from the average range of 6 to 8 to double, triple or more. It is important to distinguish between PCOS (the syndrome) and a woman with PCO (polycystic ovaries): to have PCOS, a woman must have at least two of these three symptoms (PCO, anovulation/oligoovulation and hyperandrogenism). This means that a woman can have PCOS (displaying anovulation and hyperandrogenism) without having PCO. Conversely, having PCO does not indicate that a person. Common signs and symptoms of PCOS include the following:

- Menstrual disorders: PCOS mostly produces oligomenorrhea (fewer than nine menstrual periods in a year) or amenorrhea (no menstrual periods for three or more consecutive months), but other types of menstrual disorders may also occur.
- Infertility: This generally results directly from chronic anovulation (lack of ovulation).
- High levels of masculinizing hormones: Known as hyperandrogenism, the most common signs are acne and hirsutism (male pattern of hair growth, such as on the chin or chest), but it may produce hypermenorrhea (heavy and prolonged menstrual periods), androgenic alopecia (increased hair thinning or diffuse hair loss), or other symptoms. Approximately three-quarters of women with PCOS (by the diagnostic criteria of NIH/NICHD 1990) have evidence of Hyperandrogenemia.
- Metabolic syndrome: This appears as a tendency towards central obesity and other symptoms associated with insulin resistance, including low energy levels and food cravings. Serum insulin, insulin resistance, and homocysteine levels are higher in women with PCOS.

Although 80% of PCOS presents in women with obesity, 20% of women diagnosed with the disease are non-obese or "lean" women. However, obese women that have PCOS have a higher risk of adverse outcomes, such as hypertension, insulin resistance, metabolic syndrome, and endometrial hyperplasia.

Even though most women with PCOS are overweight or obese, it is important to acknowledge that non-overweight women can also be diagnosed with PCOS. Up to 30% of women diagnosed with PCOS maintain a normal weight before and after diagnosis. "Lean" women still face the various symptoms of PCOS with the added challenges of having their symptoms properly addressed and recognized. Lean women often go undiagnosed for years, and usually are diagnosed after struggles to conceive. Lean women are likely to have a missed diagnosis of diabetes and cardiovascular disease. These women also have an increased risk of developing insulin resistance, despite not being overweight. Lean women are often taken less seriously with their diagnosis of PCOS, and also face challenges finding appropriate treatment options. This is because most treatment options are limited to approaches of losing weight and healthy dieting.<sup>[10]</sup>

**Clinical features, hormonal profile, and metabolic abnormalities of obese women with obese polycystic ovary syndrome** The data of the anthropometric measurements, clinical manifestations of hyperandrogenism, serum levels of luteinizing hormone (LH), follicle stimulating hormone (FSH), estradiol (E(2)), testosterone (T), prolactin (PRL), dehydro-epiandrosterone sulfate (DHEAS), sex-hormone-binding globulin (SGBG), and 17-oxyhydroprogesterone (17-OHP), fasting plasma glucose (FPG) and fasting insulin (FINS) detected after oral glucose tolerance test (OGTT), serum lipid levels, including total cholesterol (Chol), triglycerides (TG), high-density lipoprotein (HDL), and low-density lipoprotein (LDL), homeostasis model assessment (HOMA) and area under curve (AUC) so as to assess the insulin resistance (IR), free androgen index (FAI) to estimate the extent of hyperandrogenism, HOMA IS and DeltaI(30)/DeltaG(30) used to assess the function of islet beta cells, were collected from 192 women with PCOS, aged 24 +/- 6, that were divided into 2 groups according to the body mass index (BMI): Group A (n = 70) with the BMI > or = 25 kg.m(-2) and Group B (n = 122) with the BMI < 25 kg.m(-2), and 65 age-matched bilateral tubal block factor infertile women served as controls that were divided into 2 groups as well: Group C (n = 25) with the BMI > or = 25 kg.m(-2); and Group D (n = 79) with the BMI < 25 kg.m(-2), and underwent a cross-sectional study.<sup>[12]</sup>

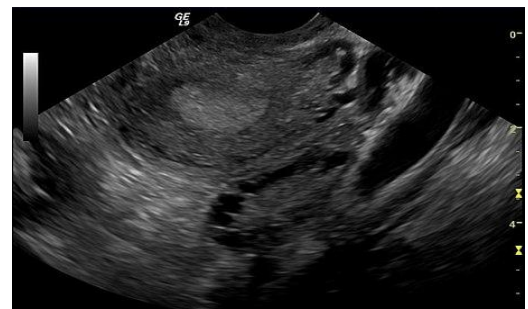
#### V. DIAGNOSIS :

Not everyone with PCOS has polycystic ovaries (PCO), nor does everyone with ovarian cysts have PCOS; although a pelvic ultrasound is a major diagnostic tool, it is not the only one. The diagnosis is fairly straightforward using the Rotterdam criteria, even when the syndrome is associated with a wide range of symptom

Fig No: 4 Polycystic ovary as seen on sonography



Fig No: 5 Transverse section of polycyst ovary



There's no test to definitively diagnose PCOS. Your doctor is likely to start with a discussion of your medical history, including your menstrual periods and weight changes. A physical exam will include checking for signs of excess hair growth, insulin resistance and acne. Your doctor might then recommend.

**A pelvic exam:** The doctor visually and manually inspects your reproductive organs for masses, growths or other abnormalities.

**Blood tests:** Your blood may be analyzed to measure hormone levels. This testing can exclude possible causes of menstrual abnormalities or androgen excess that mimics PCOS. You might have additional blood testing to measure glucose tolerance and fasting cholesterol and triglyceride levels.

**An ultrasound:** Your doctor checks the appearance of your ovaries and the thickness of the lining of your uterus. A wand like device (transducer) is placed in your vagina (transvaginal ultrasound). The transducer emits sound waves that are translated into images on a computer screen. If you have a diagnosis of PCOS, your doctor might recommend additional tests for complications. Those tests can include: Periodic checks of blood pressure, glucose tolerance, and cholesterol and triglyceride levels. Screening for depression and anxiety Screening for obstructive sleep apnea.<sup>[11]</sup>

#### VI. Treatment:

PCOS treatment focuses on managing your individual concerns, such as infertility, hirsutism, acne or obesity. Specific treatment might involve lifestyle changes or medication.

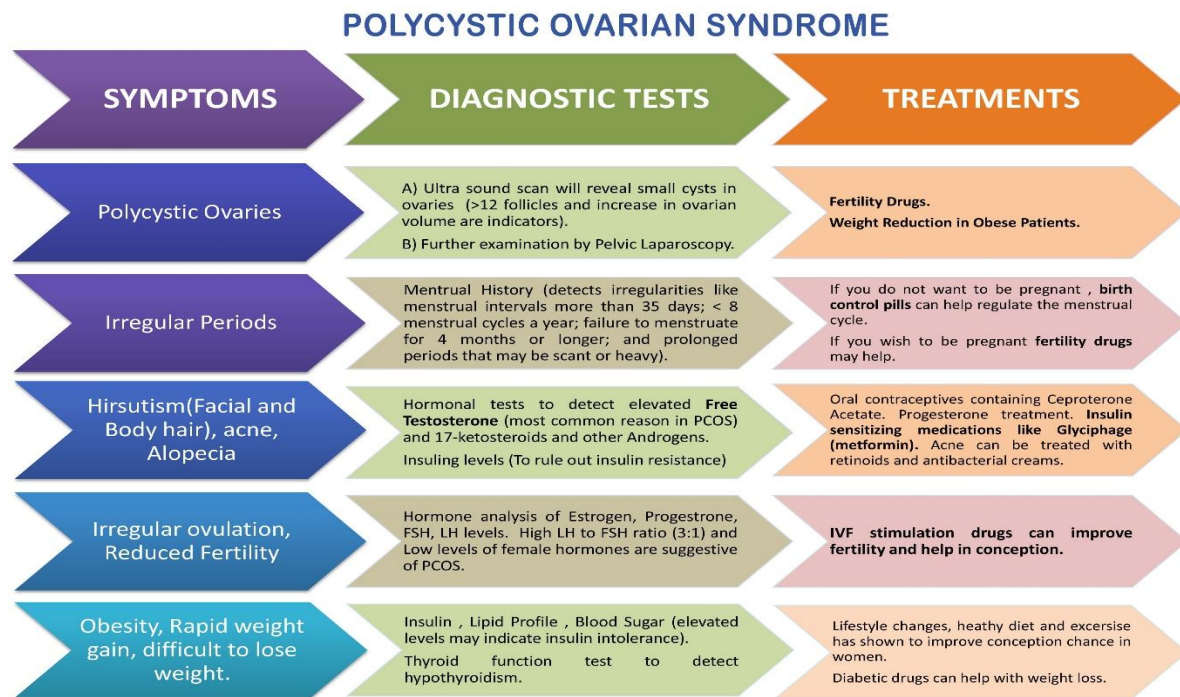


Fig No: 6 PCOS Symptoms, Diagnosis, Treatment.

**Lifestyle changes**

Your doctor may recommend weight loss through a low-calorie diet combined with moderate exercise activities. Even a modest reduction in your weight — for example, losing 5 percent of your body weight — might improve your condition. Losing weight may also increase the effectiveness of medications your doctor recommends for PCOS, and can help with infertility.

**Medication:**

To regulate your menstrual cycle, your doctor might recommend:

**Combination birth control pills:** Pills that contain estrogen and progestin decrease androgen production and regulate estrogen. Regulating your hormones can lower your risk of endometrial cancer and correct abnormal bleeding, excess hair growth and acne. Instead of pills, you might use a skin patch or vaginal ring that contains a combination of estrogen and progestin.

**Progestin therapy:** Taking progestin for 10 to 14 days every one to two months can regulate your periods and protect against endometrial cancer. Progestin therapy doesn't improve androgen levels and won't prevent pregnancy. The progestin-only minipill or progestin-containing intrauterine device is a better choice if you also wish to avoid pregnancy.

To help you ovulate, your doctor might recommend:

**Clomiphene:** This oral anti-estrogen medication is taken during the first part of your menstrual cycle.

**Letrozole (Femara):** This breast cancer treatment can work to stimulate the ovaries.

**Metformin:** This oral medication for type 2 diabetes improves insulin resistance and lowers insulin levels. If you don't become pregnant using clomiphene, your doctor might recommend adding metformin. If you have prediabetes, metformin can also slow the progression to type 2 diabetes and help with weight loss.

**Gonadotropins:** These hormone medications are given by injection.

**Birth control pills:** These pills decrease androgen production that can cause excessive hair growth. Birth control pills helps in controlling the menstrual cycles, lower the male hormones which pose a block in ovulation and also help to clear acne. It also helps in clearing the ovaries from the remaining tiny under-matured follicle staying in the ovaries as cysts. Thus, many physicians prescribe birth control pills for a few months to women who are willing to conceive so that the ovaries are cleared and the menstrual cycles come to normal which in turn better promotes the process of conception. A physician might also prescribe only Progesterone hormone pills to regulate the menstrual cycle instead of contraceptive pills. The purpose served is same for both.

**Spirolactone (Aldactone):** This medication blocks the effects of androgen on the skin. Spirolactone can cause birth defects, so effective contraception is required while taking this medication. It isn't recommended if you're pregnant or planning to become pregnant.

**Eflornithine (Vaniqa):** This cream can slow facial hair growth in women.

**Electrolysis:** A tiny needle is inserted into each hair follicle. The needle emits a pulse of electric current to damage and eventually destroy the follicle. You might need multiple treatments.

**Maintain a healthy weight:** Weight loss can reduce insulin and androgen levels and may restore ovulation. Ask your doctor about a weight-control program, and meet regularly with a dietitian for help in reaching weight-loss goals.

**Limit carbohydrates:** Low-fat, high-carbohydrate diets might increase insulin levels. Ask your doctor about a low-carbohydrate diet if you have PCOS. Choose complex carbohydrates, which raise your blood sugar levels more slowly.

**Be active:** Exercise helps lower blood sugar levels. If you have PCOS, increasing your daily activity and participating in a regular exercise program may treat or even prevent insulin resistance and help you keep your weight under control and avoid developing Diabetes

**Treatment for Polycystic Ovarian Syndrome to Restore Fertility:** Polycystic ovarian syndrome though cannot be cured completely due to its genetic base for some; it could be managed very easily. Depending on the focus of treatment or the goal, the treatment plan may vary.

**Medications to Induce Ovulation and Boost Fertility:** Anovulation is the basic cause for women with PCOS to not conceive. Thus, medications can be prescribed to induce and stimulate ovulation, maturation and rupturing of the follicle. The types of medication prescribed are Clomiphene, Metformin along with clomiphene and Gonadotrophin. Gonadotrophins are generally available as injection and are much expensive. This is generally given depending upon the hormonal levels and also for those who are not reacting optimally to the oral drugs. However, fertility medications raise the risk of multiple births as they tend to stimulate more than one follicle which if ruptured and fertilized can result in twins and triplets.

**Weight Loss:** Although the fact that either obesity induces PCOS or PCOS increases obesity is not yet established statistically, yet researchers have confirmed that 50% of women with PCOS are obese. Moreover, studies have also confirmed that even a 5% loss in weight can reduce the androgen levels in the body by 20 percent. The dropping down of androgen levels helps in enhancing ovulation and regularizing the menstrual cycle. Studies also suggest that the 60% of women who could achieve ovulation by losing weight have higher chances of conception in the following 3-4 months. Thus weight loss and management is very important in boosting your fertility.

**Assisted Reproductive Technologies to Aid Fertility Problems:** If the above treatments fail to give a pregnancy, these women have the boon of advanced technologies like ovulation induction, Intrauterine Insemination (IUI), In-vitro Fertilization (IVF) etc<sup>[9]</sup>.

#### CONCLUSION:

PCOS is a common disorder of women that is associated with significant reproductive and nonreproductive morbidity as outlined here. Perception of this and preventative therapies are important for the health care of women. For PCOS, diet, exercise, and oral contraceptives are reasonable preventative therapies. Screening for hypertension, abnormal lipid profiles, insulin resistance, and reproductive disorders including cancer should be the mainstay of care for women with PCOS.

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