**JCRT.ORG** 

ISSN: 2320-2882



# INTERNATIONAL JOURNAL OF CREATIVE **RESEARCH THOUGHTS (IJCRT)**

An International Open Access, Peer-reviewed, Refereed Journal

# **Pcos: AN OVERVIEW OF LIFESTYLE** DISORDER FOUND OF IN 90% FEMALES IN WORLDWIDE.

Aarti Sawant, Naziya Sayyad\*, Snehal Ugale, Dr. Mahendra Gaikwad, Tejaswini Kande.

#### Abstract: -

Polycystic ovary syndrome is a heterogeneous disorder. It is caused due to chronic anovulation. The symptoms of this PCOS disease occurs during the puberty years. Both normal female pubertal development and pcos are characterized by irregular menstrual cycle, anavulation and acne. Olegomenorrhea (less than 9 period for year) and Amenorrhea (no menustrual period for at least six months) are features of PCOS and can be regarded as' 'as risk for PCOS'.so there are some medication for management of PCOS disease such as progestin therapy, spironalactone, clomiphene, letrozole, Gonadotropins, Eflornithin.

# Keywords: -

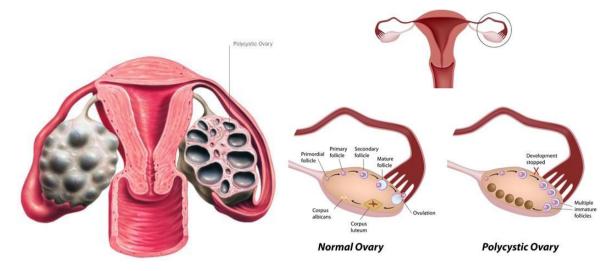
Menstrual Cycle, Anovulation, Olegomenorrhea, Amenorrhea, Hyperandrogonism.

#### **INTRODUCTION: -**

Polycystic ovaries syndrome is hormonal imbalance in which ovaries (female reproductive organ) produce higher than normal level of testosterone (the male sexual hormone). This imbalance can results in irregular menstrual cycle and in the some cases, infertility or inability to bear children. PCOS affect the women of age group 14 to 40.It may involve the combination of genetic and environmental factors. Ovaries may develope cysts -Women diagnosed with the PCOS has three times risk of diabetes, stroke and heart disease. Twice risk of anxiety depression and drugs use Twice risk of hospitalisation. Ten times risk of infertility.

#### What is PCOS?

A Hormonal disorder causing enlarge ovaries with small cyst on the outer edges. It happens during the reproductive years. With PCOS, many small sacs of fluid develope along the outer edges of ovary. These are called cyst .The small fluid filled cysts contain immature eggs and called as follicles. The follicles fail to regular release of eggs but the exact causes of PCOS are unknown.



# **Etiology:**

PCOS is a oligogenic disorder. In this disorder, there is interaction of 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 is relatively common however familial links to PCOS are unclear. A lack of phenotypic information prevent the formal segregation analysis. But the current literature suggest that, the clustering of PCOS in families resembles an autosomal dominant pattern. Environmental factor implicated in PCOS (eg. Obsity) can be exacerbated by poor dietary choice and physical inactivity. The agents which cause infection and also toxins play role. The the reproductive and metabolic features of PCOS are sometime reversible with lifestyle modification such as weight loss and exercise.

#### **Risk factors:**

Risk factor of PCOS in adults include type 1 diabetes, type 2 diabetes and gestational diabetes. Insulin resistance affect the 50% to 70% women with PCOS leading to number of comorbidities including the metabolic syndrome, hypertension, dyslipidemi ,glucose, intolerance and diabetes. The genetic components are also risk factor for PCOS. Certain changes in utero main affect PCOS before the born, mood problem such as depression, anxiety and physiological stress are cause PCOS. Obsity history of eclipsy are also risk for PCOS.

## Clinical aspects of PCOS and characteristics:

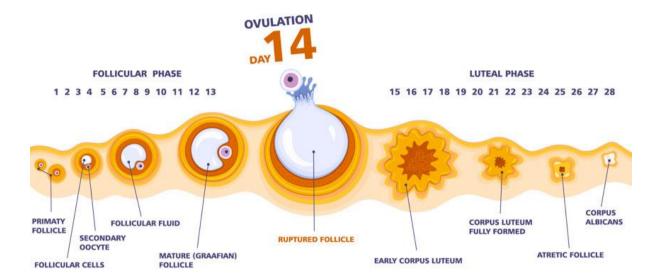
Women with PCOS can present with diverse symptoms, include irregular menstrual cycle, excessive hair growth, subfertility and pregnancy complication. It includes the lifestyle changes, medication and sometimes even the bariatric surgery for prevention and management of obesity. Goals of therapy include, fertility, decreased hirustism or alopecia and providing endometrial protection in order to avoid endometrial Cancer. Early identification of high risk patients enables through the prevention screening and early treatment of adverse complication. At least four clinical phenotypes have been dalinated according to current diagnostic criteria.

## **PCOS** symptoms:

#### 1. Menstrual irregulaty:

- •olegomenorrhea:-less than 9 menstrual periods per year.
- -menstruation interval 35 days or greater
- •Amenorrhea: -secondary amenorrhea
- no menstrual period for at least 6 month.

# **MENSTRUAL CYCLE**



# 2. Infertility:

- due to menstrual irregularities
- -Chronic anavolution
- -risk of miscarriage is also increase.

# 3. Polycystic ovaries : -

due to anovulution -multiple cysts within the ovaries.

- enlarged ovaries.

# 4. Acne vulgaris:

- due to elevated androgen level.



# 5. Hirustism:

- increase growth of terminal hairs
- face ,upper lip, chin, neck ,lower abdomen.
- due to elevated androgen level.





# 6. Alopecia:

- -Hair thinning
- -male pattern baldness
- due to increased level of androgen.

# 7. Hyperthecosis:

- -Server case of PCOS.
- very high level of androgen cause increased masculinization

# **Noticeable symptoms of PCOS:**

- unpredictable and irregular periods.
- acne
- -course hair developing on
- face
- chest
- Belly •back
- Thick dark skin developing on neck, underarms, grain (acanthosis nigicans) -depression and other mood disorder.

## **Hidden Symptoms of PCOS:**

- -Risking blood sugar leading to pre diabetes and diabetes
- High blood pressure
- High cholesterol levels.
- Sleep apnea (snoring and daytime sleepiness)
- risk of cancer such as uterine cancer.

#### **Investigation:**

The most common endocrine and metabolic disorder in Pre menopausal women is PCOS, characterized by hyper androgenism, chronic anovulation on ultrasound evidence of small ovarian cysts . Testosterone, androstenedione or LH either alone or in combination where rised in 86% of women with PCOS and these should be definitive hormonal tests. Using criterion for diagnosis for PCOS should be abandoned because of its low sensitivity. Blood tests can measure hormone levels. This testing exclude possible causes of menstrual problem or androgen excess that mimic PCOS. Investigation performed majority more than 80% of time included gonadotropins (LH and FSH), dehyadroepiandrosterone sulphate, free testosterone and 17 hydroxyprogesteron. Fewer than one half of adolescent 17 of 36(47%) had an LH to FSH ratio greater than 2.Lipid such as cholesterol and triglycerides were collected in approximately one half (21-41) of adolescents. Combined hyperlipidemia was present in 12 of 21

adolescent (57%).HLD and low density lipoprotein cholesterol level available in 17&19 of 41 girls and we're abnormal in 2 girls.

Laboratory test	Total number screened, n (%)*	Adolescents sc		
		Endocrinology (E) (n=21)	Gynecology (G) (n=20)	χ <sup>2</sup> P (E versus G)
LH	36 (87.8)	16 (76.2)	20 (100)	0.02
FSH	36 (87.8)	16 (76.2)	20 (100)	0.02
DHEAS	35 (85.4)	20 (95.2)	15 (75.0)	0.07
Androstenedione	22 (53.7)	16 (76.2)	6 (30.0)	0.003
Testosterone	18 (43.9)	12 (57.1)	6 (30.0)	0.08
Free testosterone	35 (85.4)	19 (90.5)	16 (80.0)	0.34
Triglycerides	22 (53.7)	8 (38.1)	14 (70.0)	$0.04^{\dagger}$
Qelesterol	21 (51.2)	8 (38.1)	13 (65.0)	0.08

Mean serum values of biochemical investigations performed and percentage of adolescents with abnormal values *							
Laboratory test	Normal value	Adolescents screened (n=41), n	Mean serum value (± SD)	Abnormal test result, n (%)			
Serum gonadotrop	oins						
LH (IU/L)	8–12 years of age: 2– 13; >12 years: <25	36	12.4 (6.49)	5 (13.8)			
FSH (IU/L)	<13	36	5.81 (1.74)	0 (0)			
Androgens							
DHEAS (µmol/L)	10–12 years of age: <8; 13–19 years of age: 1–12	35	6.85 (2.81)	1 (2.9)			
Androstenedione (Qnol/L)	1.7–7	22	7.23 (2.90) <sup>†</sup>	9 (40.9)			
O' arono_	07_24	10	_252	0 (50)			



Mean serum values of biochemical investigation with abnormal values. Approximate two thirds (28 of 41) 68% of adolescent who met Canadian Diabetes Association criteria for T2DM testing were screened. Canadian Diabetes Association criteria for FBG or OGTT screening include individuals who are overweight and have two other risk factors. Pelvic ultrasound was performed in 28 adolescent (68%)

Approximate 15% (4 of 28) of those adolescent who underwent imaging shows evidence of ovarian cyst consistent with diagnosis for PCOS. There were no adolescent detected with congenital adrenal hyperplasia, hyperprolactinemia or thyroid dysfunction.

## **Management:**

- -lifestyle changes -ovulation indication -metformin (insulin sensitizer)
- Assisted Reproductive Technologies (ART): IVF, ICSI, IVM
- laparoscopic ovarian drilling
- -bariatric surgery
- inocitol

#### **Recent advance in pcos:**

New genetic research suggest men can develop characteristics of PCOS- common metabolic and reproductive disorder than affects the women. Diagnostic criteria of PCOS which remain most used world wild for both individual diagnosis research It defined the PCOS as presence of any two of three features: hyper androgenation. ovulation disfunction and •Polycystic ovarian morphology by ultrasound. Recent guideline from international PCOS network recommended used of Rotterdam criteria in adults and requirements of both oligoanovulation and hyperandragonism of PCOS diagnosis in adolescents. To better understand pathogenesis of syndrome, it is important to compare the getetic profiles of women whose diagnosis was based on different phenotypes. A Recent genome - wide meta analysis from over 10,000 PCOS cases identified 14 independence loci associated with risk of PCOS including, 3 novel loci. Anti mullarian hormone (AMH) is glycoprotein secreted by granulosa cells of pre-antral and small antral follicles. AMH plays an essential role in sexual differentiation and gonadal function , besides Central effects on hypothalamic pituitary gonadal axis. AMH receptor is expressed in Gondatropin releasing hormone (GnRH)neurones and that intracerebroventricular administration of AMH increased GnRH dependent LH pulsatile realease. There is accumulating evidence that GnRH pulsetility is pulsatility is perturbed in women with PCOS, leading to increased LH Pulsastility, which plays important role in PCOS pathophysiology. Serum AMH levels are typically increased in PCOS and therefor AMH dependent regulation of GnRH release could be involved in path of pathophysiology of fertility in women with PCOS.

# **Ongoing clinical trials on PCOS:**

Ayurveda works with the vision of providing healthy and stress free life to people. It uses natural herbs and medicinal herbs to treat the disease. for PCOS certain spices like ashwagandha and turmeric are used with increase the yoga and breathing exercise. In PCOD, Ayurveda uses therapies to ensured cyst dissolution gandhari and Varuna are major herbs that promotes ovulation and cyst dissolution. Also it helps to build up the metabolism and keep the gynecological disorder away.

# **New research and development:**

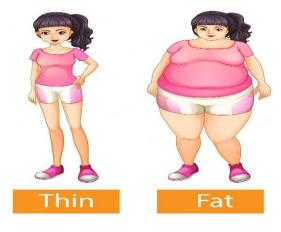
In normal women, androgen production rate (PR)is results of adrenal, ovarian secretion and conservation from precursor's in peripheral tissues, particular adipose tissue and skin.

Similarly, MCR (metabolic clearance rate) occurs in both glandular and extraglandular tissues. Both PR and MCR of androgeneous in female depends on age. In addition, it was higher in obese PCOD women and varried according to its PR where as MCR of androstenedion was marginally different with respect to normal weight affected women. Estrogen and progesterone PRs in women with PCOD investigated.

# **Treatment:**

PCOS treatment focuses on managing the infertility, hirsutism, acne or obesity.

- lifestyle changes:
- -losing the weight through the low calorie diet might improve the condition.
- It increase effectiveness for PCOS and help with infertility. Positive attitude, self confidence, self motivation for redution of weight is imortant.



- Medication:
- Combination birth control pill
- Progestin therapy
- Clomiphene



- latrozole(famara)

- -Metformin
- Gonadotropis
- -Spironolactone (Alsactone)
- -Eflornithine (vaniga)

## Significance of adrenal adrogen production:

25% of adrenostenedione and testosterone production is of ovarian origin.

In women androgenes serves as precursors of estrogen biosynthesis, which starts to decrease 3 to 4 years before menopause.

#### Reference:

- 1. Rotterdam ESHRE/ASRM-Sponsored PCOS consensus workshop group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to Polycystic Ovary Syndrome (PCOS) Human Reproduction. 2004;19:41-47.
- 2. Azziz R, Carmina E, Dewailly, et al. Criteria for defining polycystic ovary syndrome as a predominantly hyperandrogenic syndrome: an androgen excess society guideline. Journal of Clinical Endocrinology & Metabolism. 2006;91:4237-4245.
- 3. Escobar-Morreale HF, Botella-Carretero JI, Alvarez-Blasco F, et al. The polycystic ovary syndrome associated with morbid obesity may resolve after weight loss induced by bariatric surgery.

Journal of Clinical Endocrinology & Metabolism. 2005;90:6364–63

- 4. Dunaif ACR, Franks S, Legro RS, editors. Polycystic Ovary Syndrome: Current Controversies, from the Ovary to the Pancreas. Humana Press; 2008.
- 5. Longcope C. Adrenal and gonadal androgen secretion in normal females. Clinical Endocrinology & Metabolism. 1986:15:213-228.
- 6. Bardin CW, Lipsett MB. Testosterone and androstenedione blood production rates in normal women and women with idiopathic hirsutism or polycystic ovaries. Journal of Clinical Investigation. 1967;46:891–902.
- 7. Pasquali R, Casimirri F. The impact of obesity on hyperandrogenism and polycystic ovary syndrome in premenopausal women. [Review] Clinical Endocrinology (Oxford) 1993;39:1–16. ]
- 8. Rosner W, Auchus RJ, Azziz R, et al. Utility, Limitations, and Pitfalls in Measuring Testosterone: An Endocrine Society Position Statement. Journal of Clinical Endocrinology and Metabolism. 2007;92:405–413.
- 9. Stener-Victorin E, Holm G, Labrie F, et al. Are there any sensitive and specific sex steroid markers for polycystic ovary syndrome? Journal of Clinical Endocrinology and Metabolism. 2010;95:810–819.