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CURRENT ASPECTS OF POLYCYSTIC OVARIAN SYNDROME: A REVIEW

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ABSTRACT

Polycystic ovary syndrome (PCOS) is one of the most common endocrine and metabolic disorders in premenopausal females. PCOS is heterogenous in nature, defined by a combination of hyperandrogenism and signs of ovarian dysfunction and the syndrome, with no other specific diagnosis. The pathophysiological aspect of PCOS focuses primarily on hyperandrogenism, which leads to hormonal dysfunction, insulin resistance, and impaired follicular formation. With risk of associated comorbidities such as endometrial cancer and type II diabetes. This review summarizes the most relevant and up-to-date reports on PCOS, briefly address the pathophysiology of the disease, and then detail its diagnostic criteria. Throughout the review, we highlight the complexity of PCOS in terms of pathophysiology, diagnosis, and the required interdisciplinary therapeutic approach.

KEYWORDS: Polycystic ovary syndrome, Hyperandrogenism, Anovulation, Hirsutism, Insulin resistance etc.

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is one of the most common gynaecological endocrine disorders in women of childbearing age.[1] It is associated with reproductive, metabolic, and mental disorders. Overall, 6% to 8% of women of childbearing age suffer from PCOS, making PCOS one of the most common endocrine disorders.[2] The name polycystic ovary syndrome refers to the large number of small cysts (fluid-filled cysts) that form in the ovaries.[3] Polycystic ovary contains a large number of harmless follicles measuring 8 mm. A follicle is an underdeveloped sac in which an egg develops. In PCOS, these pouches often cannot release eggs, which mean that ovulation does not occur.[4] Polycystic ovary syndrome (PCOS) was first described in modern medical literature by Stein and Leventhal in 1935, describing seven women suffering from amenorrhea, hirsutism, and ovarian enlargement. Also known as Stein Leventhal Syndrome.[5] However, women with PCOS are at risk of developing pregnancy-related complications such as gestational diabetes, endometrial cancer, and infertility such as mental illness.[6] PCOS has been diagnosed with hyperandrogenism, anovulation, amenorrhea or amenorrhea, and cysts of various sizes in the ovaries, but there are significant differences between patients.[7]

Little is known about the aetiology of this syndrome, but increased evidence suggests that PCOS may be a complex epigenetic disease with strong epigenetic and environmental consequences doing. In recent years, with the help of high-resolution ultrasonography of the ovaries, PCOS has been found to be a heterogeneous disease with a spectrum of clinical and biochemical features.[8] Through a review of, we highlight the complexity of PCOS in terms of pathogenesis, pathophysiology, clinical signs and symptoms, diagnostics, and therapeutic approaches.[9]

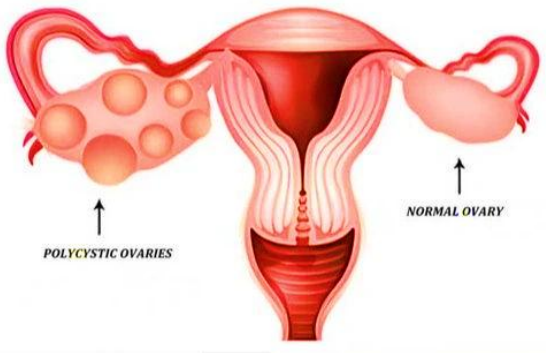

Fig. 1: Polycystic ovary and Normal ovary.

Etiology

The basic cause of PCOS is still unknown, and the molecular basis for its progression is still a mystery. In general, genetic and environmental factors are involved in the etiology of this disease. An unhealthy lifestyle, an unhealthy diet, or an ant-infected mediator increases the

risk of PCOS. Hyperinsulinemia is a major cause of excess androgens because insulin directly stimulates the effects of LH and indirectly increases GnRH. Insulin lowers sex hormone-binding globulin (SHBG), the major circulating protein that regulates testosterone levels. When SHBG decreases, free androgens increase, causing clinical symptoms such as hirsutism, alopecia, and acne. [10] The role of IR and hyperinsulinemia in the development of PCOS has been extensively studied and is widely recognized to play an important role in the molecular mechanisms involved in androgenic hypersecretion typical of this condition. [11]

In addition to genetic and environmental factors, other factors such as obesity, ovarian dysfunction, and hypothalamic-pituitary abnormalities all contribute to the pathogenesis of PCOS. Exclusions of related disorders such as hyperprolactinemia, thyroid disease, and congenital adrenal hyperplasia include etiology.

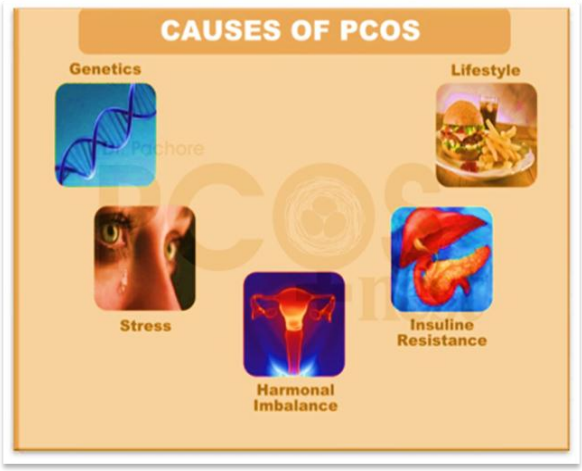

Fig. 2: Etiology of PCOS.

Pathophysiology

PCOS is characterized by excessive ovarian and / or adrenal androgen secretion. Endogenous ovarian factors such as altered steroid production and extraovarian factors such as hyperinsulinemia contribute to excess ovarian androgen production. [12] Most commonly, insulin resistance and compensatory hyperinsulinemia are associated with hyperandrogenism by stimulating androgen secretion in the ovaries and inhibiting the production of sex hormone-binding globulin in the liver.[13] Normally, 80% of testosterone binds to sex hormone binding globulin, 19% binds to albumin, and only 1% circulates as free testosterone. The concentration of SHBG helps to calculate the free androgen index, which helps determine the level of the hormone (testosterone) in men in the blood. [14]

Testosterone is an important hormone that contributes to the pathophysiology of PCOS. It is detected by increased levels of free or unbound testosterone in the bloodstream. [10] Hirsutism and hyperandrogenism are signs of excessive androgen production. IR or hyperinsulinemia results in increased IGFI (insulin-like growth factor) levels by inhibiting the production of IGFBPI (IGF binding protein I) and increasing LH-stimulated androgen synthesis. Elevated androgen levels contribute to the cause of anovulation and polycystic ovary. [15]Genetic predispositions such as the gonadotropin gene and the diabetic gene are also involved in the pathophysiology of PCOS. The exact mechanism by which PCOS develops is not yet fully understood, but the abnormal functioning of women with PCOS has several characteristics.

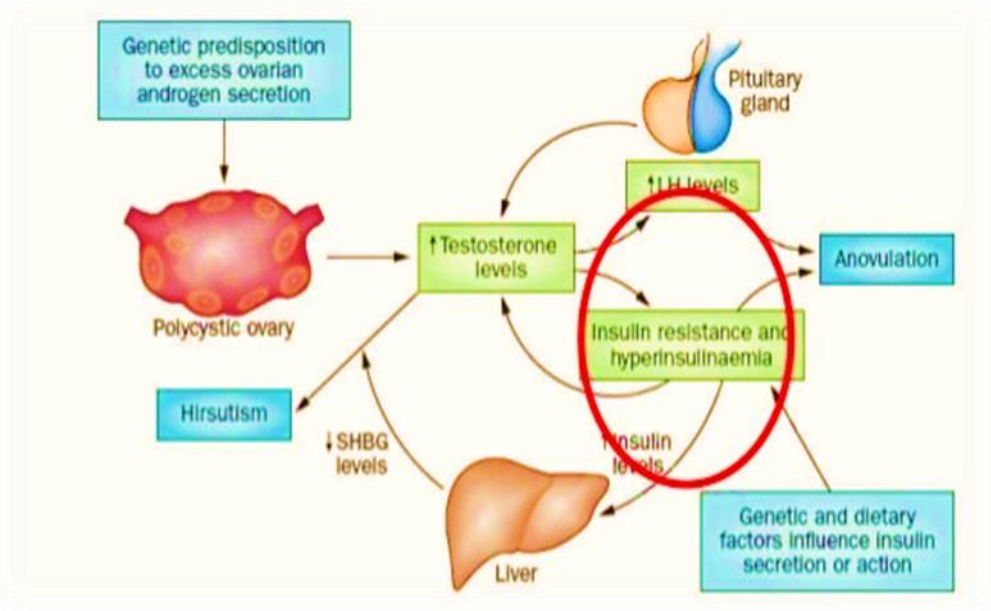

Fig. 3: Pathophysiology of PCOS.

Common Signs and Symptoms of Pcos
Menstrual Disturbances[1]

- Oligomenorrhea (Infrequent periods)
- Amenorrhea (Absence of periods)
- Difficulty conceiving or infertility or recurrent miscarriages

Hyperandrogenism[4]

- Hirsutism – Excessive hair growth on face and body
- Cystic acne
- Male pattern alopecia (Baldness on head)

Other

- Obesity/weight gain/inability to lose weight
- Obstructive sleep apnea
- Acanthosis nigricans (black or brown hyperpigmentation) on dermal areas, especially behind neck and in skin folds
- Episodes of depression
- Galactorrhea and elevated prolactin levels
- Hyperlipidemia and hypertension
- Pelvic pain
- More than 12 cysts in one ovary on ultrasound appearance
- Mood disorders
- Increased craving for carbohydrates

PCOS DIAGNOSIS

Doctors can diagnose PCOS by performing the following tests:

1. Physical examination -This includes measuring weight, height, blood pressure and checking for unwanted hair growth.
2. Pelvic Exam-The doctor will check for swelling and growth in the pelvic area.
3. Blood tests -Blood tests may be done to measure the levels of hormones. This includes androgen tests, thyroid tests, cholesterol and triglyceride tests, and blood glucose levels.
4. Ultrasound -When imaging of polycystic ovary, transvaginal ultrasound is considered the gold standard, especially because it optimally displays the internal structure of the ovary in obese patients. Compared to transabdominal ultrasonography, it is more effective in women with PCOS in detecting the presence of polycystic ovary. This analysis was performed on the 2-3rd day of the menstrual cycle. This is done transvaginally using an 8 MHz transducer. When using transabdominal ultrasound, do not use AFC (Antral Follicle Count) as a diagnostic criterion for PCO (polycystic ovary).

Polycystic ovary syndrome was determined based on two criteria of: ovarian volume and number of follicles. Polycystic ovary syndrome occurs when (A) the volume of the ovary exceeds 10 cm3. (B) If there are 12 or more cysts with a diameter of 2-9 mm in one or both ovaries.[16]

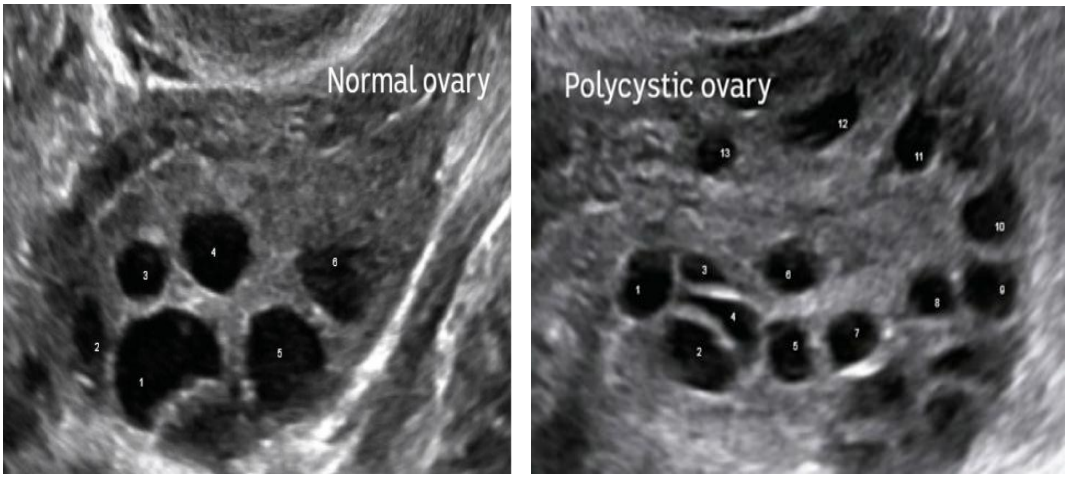

Fig. 4: Ultrasound view of Normal ovary v/s polycystic ovary.

Treatment of PCOS

Lifestyle Changes: Treatment of PCOS usually begins with lifestyle changes such as weight loss, a nutritious diet, regular exercise, and good habits.[1]

Therapeutic Treatment[17]

1. Oral contraceptives- OCP is the first-line treatment for women who do not want to ovulate and have menstrual disorders. Oral contraceptives reduce circulating androgens by increasing SHBG. It reduces the risk of ovarian cancer.
2. Clomiphene-It is used to treat infertility that causes ovulation.
3. Induction of ovulation by gonadotropins.
4. Metformin- It can effectively lower androgen levels improve insulin sensitivity and promote weight loss

in obese patients. Metformin increases glucose intake and utilization and improves insulin resistance in PCOS patients.

5. Spironolactone (Aldactone)- Spironolactone works by blocking androgenic effects. Androgens like testosterone are responsible for unwanted hair growth on the face, chest and stomach, and also increase the production of sebum on the skin, which leads to acne.
6. Pioglitazone (Actos)-This drug may lower blood insulin levels in hirsute females.
7. Rosiglitazone (Avandia) -Acts as an insulin sensitizer and also reduces excess androgens in the circulating blood.

Pharmacologic Agents for PCOS

Drug	Place in Therapy	Dosage	Adverse Effects
Oral contraceptives	Androgen excess, contraception, regulation of cycle, protection of endometrium	One tablet po daily	Breast tenderness, weight gain, fluid retention, increased risk of thromboembolism
Clomiphene	Ovulation induction	50 to 150 mg po daily days 5 to 9 of cycle	Hot flashes, nausea, headache, blurred vision, multiple gestation
Gonadotropins	Ovulation induction	Dosage and duration dependent on product and patient response	Abdominal pain, nausea, breast tenderness, injection site reaction, multiple pregnancy, ovarian hyperstimulation
Metformin (Glucophage, Glucophage XR)	Hyperinsulinemia, anovulation, androgen excess	500 mg po bid to 850 mg po tid	Nausea and vomiting, diarrhea, anorexia, metallic taste, lactic acidosis (rare)
Spironolactone (Aldactone)	Hirsutism, acne	100 to 200 mg po daily	Intermenstrual bleeding, hyperkalemia, hypotension
Pioglitazone (Actos) Rosiglitazone (Avandia)	Hyperinsulinemia, anovulation, androgen excess	30 to 45 mg po daily 4 mg po daily to bid	Weight gain, edema, increased LDL cholesterol with rosiglitazone

Fig. 5: Pharmacologic Agents for PCOS.

CONCLUSION

The review clearly demonstrates that PCOS is a complex multi-organ disorder that occurs in early puberty. It is difficult to understand and name the core mechanism. The list of factors involved in pathophysiology continues to grow, and there is increasing evidence that hyperandrogenism is a key factor affecting multiple tissues. Severity can only be reduced if the following correct precautions are taken i.e. Weight loss, healthy eating, and recommended medications.

ACKNOWELDGEMENT

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