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Review Article

POLYCYSTIC OVARIAN SYNDROME : PATHOGENESIS AND HEALTH CONSEQUENCES

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ABSTRACT:

Polycystic Ovarian Syndrome is the most common endocrinopathy ,that affects woman of reproductive age. It is characterized by excessive androgen production by the ovaries. Symptoms of PCOS may begin shortly after puberty, but can also develop during later teen years and early adulthood. It is a complex manifestation involving many factors and organs such as obesity, insulin resistance, irregular menstrual bleeding, abnormal menstrual period and cycle, anovulation. PCOS affects approximately 5 -12 % of women. Women with PCOS grow many small cyst i.e. fluid filled sacs to grow on the ovaries. That is why it is called, Polycystic Ovarian Syndrome. Women with PCOS are at higher risk of diabetes, infertility, metabolic syndrome, heart disease and high blood pressure. In *ayurveda*, this condition is not explained as a single disease but includes in *yonivyapada* (genital disorders) and *artavdushti* (menstrual disorders). *Ayurveda* considers involvement of four basic etiological factors i.e. unhealthy lifestyle, menstrual disorders, genetic defects and cryptogenic factors in the establishment of female genital disorders.

KEY WORDS: PCOS, Anovulation, Obesity, Insulin resistance, Infertility.

INTRODUCTION:

Polycystic Ovarian Syndrome is the most common endocrine disorder in a woman of reproductive age with a prevalence of 9.13% in Indian population^[1]. It is originally described in 1935 by Stein and Leventhal. It is characterized hyperandrogenism, chronic anovulation and polycystic ovaries. The incidence varies between 0.5-4 percent, more common amongst infertile women. It is prevalent in young reproductive age group (20-30%). Polycystic ovary may be seen about 20% of normal women^[2].

Organs involved in polycystic ovarian disease are^[3] –

1. Ovary – The female gonad organ present at the either side of the uterus.

2. Adrenal glands – The glands which are placed just above the both kidneys.

3. Pancreas – Glands that produces insulin in our body.

4. Pituitary Gland – The gland just below the brain, which is responsible for all the hormonal control.

Pathogenesis :

Typically the ovaries are enlarged. Ovarian volume is increased. The capsule is thickened and pearly white

in colour. Presence of multiple follicular cysts measuring about 2-9 mm in diameter are crowded around the cortex. Histologically there is thickening of tunica albuginea. Hypertrophy of theca cell. Patient may be present with features of diabetes mellitus (insulin resistance)^[4]. There is increase production of androgen by the ovaries, which suppresses the maturation of ovarian follicle. So ovum does not get properly formed and released (anovulation). Careful survey concludes that the biggest lifestyle contributor to PCOS is poor diet. Young women with PCOS tends to eat far too much sugar or carbonized drinks and highly refined carbohydrates which causes unhealthy raise in insulin levels. According to world fame Jerilyn Prior, insulin stimulates androgen receptors outside of ovary, causing typical PCOS symptoms which also play a role in blocking release of ovum from follicle. This type of diet will cause obesity and thus aggravating PCOS. Also in stressful women, as they eat more food that are high in fat, sugar and carbohydrate in response to stress, the more fat they store, thus contributing in the development of obesity linked PCOS.

PCOS may be discussed under the following heads:

- * Hypothalamic -Pituitary compartment abnormality :
 - Increased pulse frequency of GnRH leads to increased pulse frequency of LH.
 - It results in elevated level of LH and decrease FSH. This way hormonal balance is triggered in PCOD.

* Androgen excess and hirsutism :

- In some patients excessive production of androgens from ovaries and adrenal glands.
- Ovary produces excess androgen due to stimulation of theca cells by high LH.
- Adrenal glands are stimulated to produce excess androgen by stress, associated high prolactin level.
- Hyperinsulinemia also results in androgen excess.

* Anovulation :

• Because of low level of FSH, follicular growth, maturation and ovulation cannot occur inside the ovary leading to abscence of ovum production (anovulation).

* Obesity and insulin resistance :

- Obesity is recognized as an important contributory factor.
- Apart from excess production of androgen it also induces insulin resistance and hyperinsulinemia, which in turn increases androgen production.

* Long term consequences :

- Excess androgen production (mainly androstenedione) leads to thickening of the inner layer of uterus (endometrium).
- Risk of diabetes mellitus.
- Endometrial cancer.
- Dyslipidemia.
- Hypertension and cardiovascular disease.
- Atherosclerosis , Obstructive sleep apnea (obesity).

According to *Ayurvedic* view PCOS can be correlated with *Aartava kshaya* in which there is deficiency or loss of *artava*. As *artava kshaya* is a disorder involving *pitta* and *kapha doshas, medas, ambu/rasa, shukra/artava dhatu* and *ras, rakta, artav vaha strotas*. Therefore polycystic ovarian syndrome can be described with same involvement of *dosha, dhatu* and *updhatu* with *kapha* predominance manifests as increased weight, subfertility, hirsutism, diabetic tendencies and coldness. Pitta predominance manifests as hair loss, acne, painful menses, clots and heart problems. *Vata* predominance manifests with painful menses, scanty or less menstrual blood and severe menstrual irregularities^[5,6,7].

CONCLUSION :

On the basis of correlation menstrual irregularities have been described under *artava vyapadas* or *yonirogas* (uterine disorders). Obesity can be correlated with *sthaulya* which is a *santarpanjanya vikara*. Acne and baldness is correlated with *mukhdushika and khalitya*. Hyperinsulinemia results into diabetes hence can be correlated with *prameha*. In this way we can correlate the pathogenesis of PCOS according to modern and aryurveda. It results into serious health consequences which affects the life and normal healthy of patient hence proper attention must be needed.

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