OBSERVATIONAL STUDY ON CAUSATIVE FACTORS OF POLY CYSTIC OVARIAN DISEASE

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ABSTRACT:

Poly cystic ovarian disease is one of the most common endocrine metabolic disorder, characterized by hyperandrogenism, anovulation and hyperinsulinemia.The exact prevalence of PCOD is not known as the syndrome is not defined precisely. It is highly variable ranging from 2.2% to 26% globally. There are few studies conducted in South India and Maharashtra and prevalence of PCOD by Rotterdam's criteria were reported as 9.13% and 22.5% respectively[1]. WHO estimates that it affects 116 million women worldwide (3.4%women) [2].Diagnosis of PCOD suggest an increased risk of type 2 diabetes, high blood pressure, obesity, depression, miscarriages and hirsutism[3].In this present observational study 30 patients were selectedto elicit the causative factors of PCOD.Most common*Aharaja Nidana* elicited were Irregular dietary habits, *Guruguna Pradhana Ahara,Atimadhura Ahara sevana*, junk food habit, aerated cold drinks, *Ajeernashana*, *Anashana* and *Vishamashana*.*Viharaja Nidana* were *Ratrijagarana,Avyayama and Vegdharana***.**Thus we will discuss here the etiological factorsfor the manifestation of PCOD based on observational study, which will helps to understand the disease and its future management.

Keywords:*Ayurveda*, etiological factors,*Nidana*,Polycystic ovary disease.

INTRODUCTION:

PCOD, an ill-definedheterogeneous condition with a complex pathophysiology, is one of the commonest endocrine metabolic disorder affecting 6-10% of women intheir reproductive age.[4]In 1935 Stein and Leventhel described the association of enlarged cysts in ovaries with amenorrhea, infertility and hirsutism called as PCOD, one of the leading cause of infertility. The diagnostic features of PCOD are hyperandrogenism,oligo or anovulation and polycystic ovaries.But precise etiology still remains unknown. At one end of spectrum there is asymptomatic patient with a single finding of polycystic ovarian morphology on pelvic ultrasound, at the other symptoms such as obesity, hyperandrogenism, menstrual cycle disturbance and infertility may occur single or in combination[5].In *AyurvedaNidana* i.e. etiological factors considered as root cause of the disease. *Nidana-Parivarjana* i.e. to avoid etiological factor is first step to treat the disease.Hence in present observational study we aim to analyze the etiological factors of PCODto reduce incidence of this disease and their unfavorable health effects.

OBSERVATIONS:

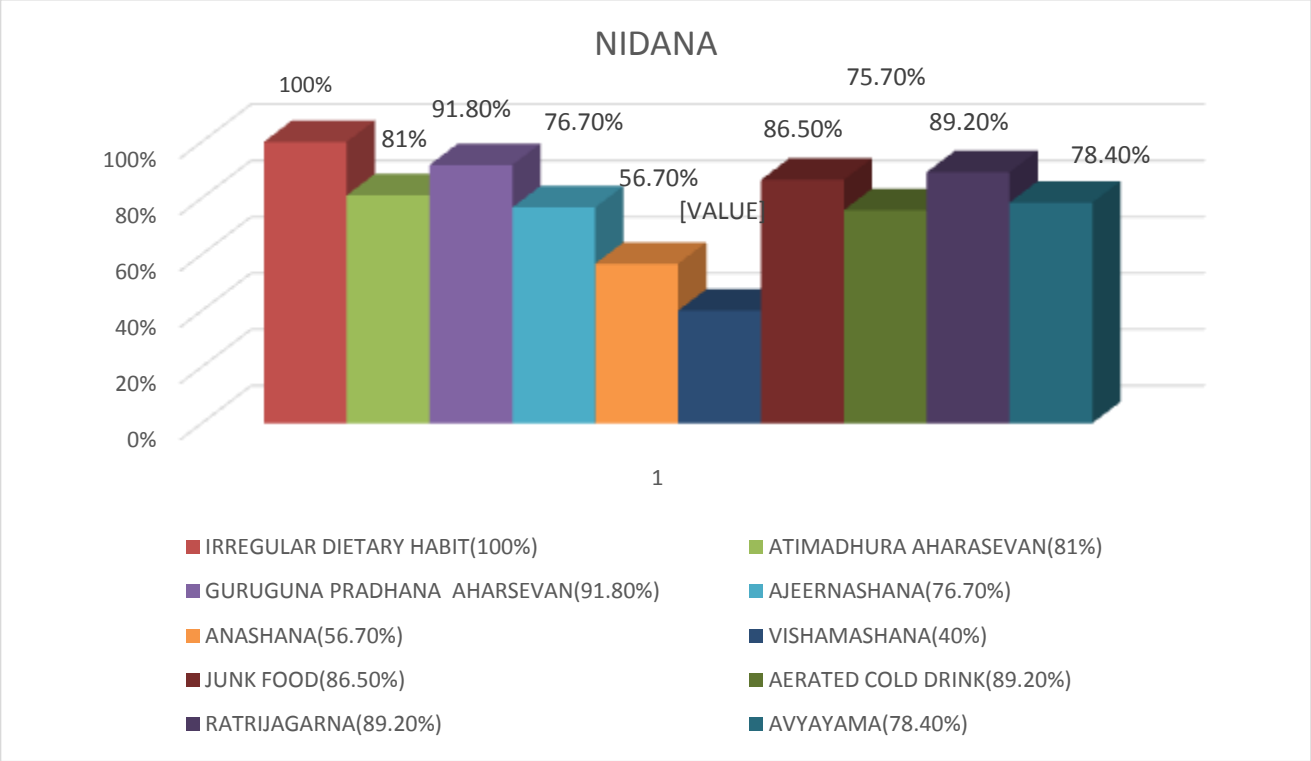

Chart No.1 Common *Nidana* seen in PCOD

Most common *Aharaja Nidana* elicited were as follow-Irregular dietary habit were found in all patients i.e. 100%.Intake of *Guruguna Pradhana Ahara* (91.8%) and *Atimadhura Aharasevana* (81%), junkfood habit (86.5%), aerated cold drinks (75.7%), *Ajeernashana* (76.7%), *Anashana* (56.7%) and *Vishamashana* (40%).*Viharaja Nidanas* were*Ratrijagarana* (89.2%),*Avyayama* (78.4%) and *Vegadharana* (29.7%).

As PCOD is caused by vitiated *Vata-Kapha*. In present study, the *Nidanas* found same *Vata-Kapha prakopaka*.

Vata- Prakopaka Nidana:

Ratrijagaranawas seen as one of the potential causative factor in 89.2% of the patients. Keeping awake late night for study and late night use of mobile phones considered as *Ratrijagarana* in this study.*Ratrou jagaranam ruksam*[5]i.e *Ratrijagrana*cause *Ruksha Guna* which cause *Vataprakopa* which was evident causative factor for PCOD.

Anashana was observed as causative factor in 56.7 % of the patients. *Anasanamalpabhojanman va*[6], in this study not taking food (*Anashana*), intake of less quantity of food (*Alpabhojana*) was considered. Due to this causative factor *Vata* gets *Prakopa* and this can be cause for PCOD.

Vegdharanawas observed in 29.7% of the patients. *Mutra*, *Shakrut* and *ArtavaNishkramana* is under the control of *Apana Vata*.When *Mutra* and *Shakrut Vegdharana* is done, it vitiates the *Apanavata* because of which *Artavpravrutti* also affected. So vitiated *Apana Vata* cause *Aniyamita Artavapravrutti* and further PCOD.

Chinta: This study shows 8.1% of the subjects were under stress. Educational stress, work stress and family stress were observed in this study. Increased stress leads to hormonal changes like raised levels of cortisol and prolactin and affects the normal menstrual cycle[7].This leads to *Vata Prakopa* and also cause *Rasavaha Sroto Dushti*, as *Artava* is *Upadhatu* of *Rasa*. Hence *Rasa Dushti* results in *Artavavaha Sroto Dushti.*

***Kapha –Prakopaka Nidana*:**

Avyayama[8]was seen as one of the potential causative factor in 78.4% of the patients.Lack of physical activity and exercise was considered as *Avyayama* in this study. It vitiated *Kapha Dosha* and further *Rasavaha* and *Medavaha Srotas* leads to *Sthaulyata .Avyayama* is also one of the cause for *Agnidushti*.This conditions were developed gradually menstrual abnormalities and PCOD.

Guruguna pradhana, Atimadhura Ahara sevana were causative factors in 91.8% and 81% of patients respectively.*Madhura rasa* has *Snigdha*,*Guru* and *Sheeta Guna* which does the *Kapha prakopa*.*Atimadhurasevana* also leads to *Sthaulya*,*Gauravata*and *Agnimandya*.This is the ultimately cause for the menstrual irregularitiesand PCOD[9].

Ajeernashana* and *Vishamashanawere causative factors in 76.7% and 40% of patients. Eating during previous meal is not digested properly is considered as *Ajeernashana*.The food taken untimely which is taken either excess or less is considered as *Vishamashana*[10].They are the main causative factors for *Agnidusti*,which further cause for Artavadushti[11] are leading to PCOD.

Irregular dietary habit in 100% which is considered as *Apathya- Ahitakara Aharasevena* which adversely affect on body and mind leads to *Tridoshaprakopa Dhatudushti-Srotodusti* and further leading to PCOD[12].

Junkfood habit 86.5%, aerated aerated cold drinks75.7%,Soda 40.5% chocolates 24.3% Chinese food 21.6%, Ice-cream 10.8% fermented food 5.4% were *Nidanas* observed in the present study.These are high in sugar fat and calories but low in nutritients,which cause obesity.High energy dense foods often lack of protein, calcium, iron, vitamin A,C,D and E,potassium, zinc and fats[13]. Nutritional deficiency leads to *Dhatukshaya* and *Vataprakopa*,which further affect the

hypothalamo-pituitary ovarian axis by disturbing the hormonal levels leads to menstrual abnormalities leading to PCOD.

Modern view on etiological factors of PCOD are as follows:

1. **GENETIC ASPECTS OF PCOD**:

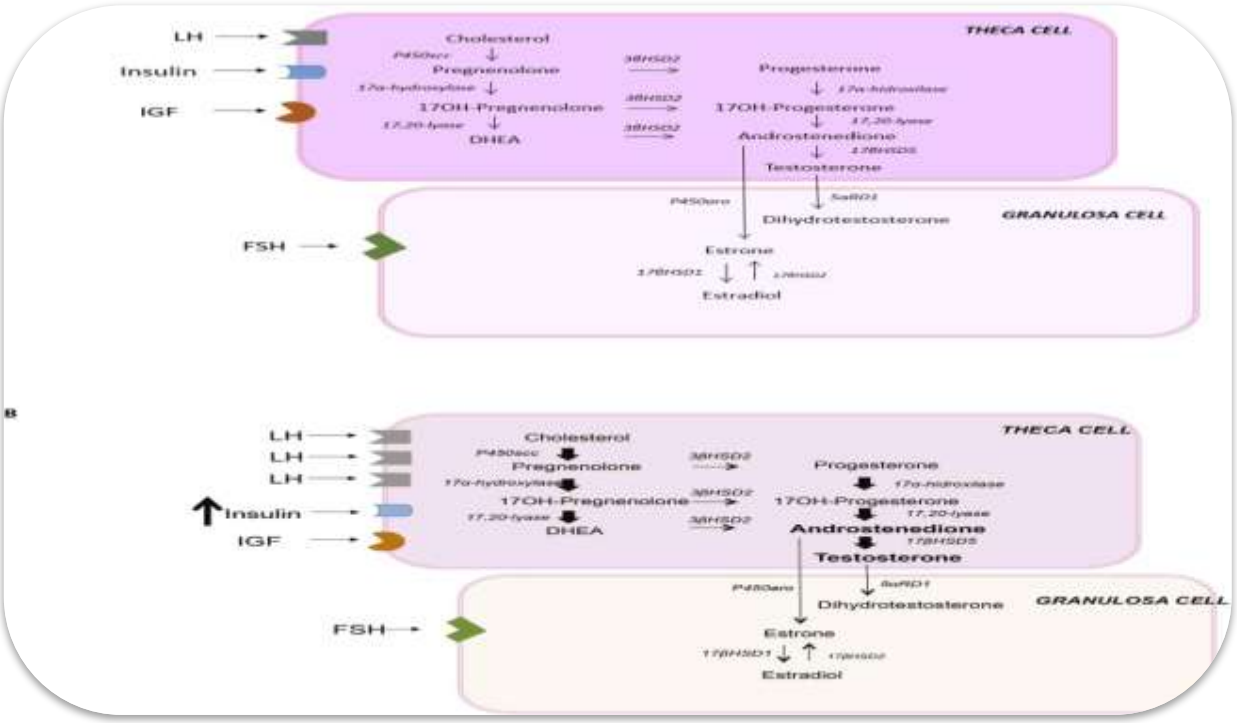

Figure1. Ovarian steroidogenesis[14] (A) Normal ovarian steroid synthesis. (B) PCOD ovarian steroid synthesis.

The most relevant genes involved in PCOS are CYP17A1, CYP11a, CYP21, CYP19, HSD3B and VNTR. (1)In comparison to normal theca cells, PCOD theca cells show increased expression of LH receptor and increased expression of CYP17A1 gene, leading to enhance of 17α-hydroxylase and 17,20-lyase activity, and amplifying androgen synthesis. (2) P450C17 enzyme activity and expression increase in ovarian theca cells in women with PCOD. (3)CYP19 converts androgens to estrogen. Aromatization deficiencyleads to hyperandrogenism,

decrease the follicular development and anovulationoccur. (4)The INS VNTR (Variable number of tandem repeats) increasesthe risk of PCOD as it develops hyperinsulinemia and menstrual disturbance.

ABNORMAL GONADOTROPIN AND STERIOD HORMONES[15]:

Hyperandrogenism:The principal sources of androgens are as follows (1)**Ovary**produces excess androgens due to stimulation of theca cell by high LH,P450C17 enzyme hyper function, defective aromatization of androgen to estrogen and stimulation of theca cells by IGF-1. (2)**Adrenals** stimulated to produce excess androgens by stress P450 C17 enzyme hyper function and associated high level of prolactin.(3)**Systemic Metabolic Alteration**: Hyperinsulinemia causes stimulation of theca cells to produce more androgens. Insulin results in morefree IGF-1 by autocrine action, IGF-1 stimulates theca cells to produce more androgens.Insulin inhibits hepatic synthesis of SHBG, resulting in more free level of androgens.

Hypothalamic Pituitary compartment abnormality:GnRHis preferential to LH rather than FSH.Increase pulse frequency and amplitude of LH results in tonicallyelevated level of LH but FSH level is not increased due to the negative feedback effect of chronically elevated estrogen and the follicular inhibin.Free estradiolincrease due to reduced SHBG bears positive feedback relationship to LH. The LH:FSH ratio is increased(3:1). Because of low FSH level, follicular growth is arrested at different phase of maturation. LH level is tonical elevated without any surge leads to Anovulation.

Inhibin: Inhibin is responsible for regulation of FSH secretion.The increase in FSH concentration can be suppressed by the release of inhibin. Women with

PCOD possess high inhibin level than normal and develop follicular atresia in ovary.

AMH: AMH has an inhibitory effect on initial recruitment of primary follicles, on FSH-dependent follicular maturation and selection of dominant follicle, and on FSH-induced aromatase expression on granulosa cells, reducing the conversion of testosterone into estradiol. Higher AMH levels in PCOD patients turns the follicles more resistant to FSH action, culminating in inhibition of follicular maturation and ovulation, and in inhibition of aromatase expression, and consequently, leading to hyperandrogenism.This advocates a significant role of this hormone in the pathophysiology of PCOD.

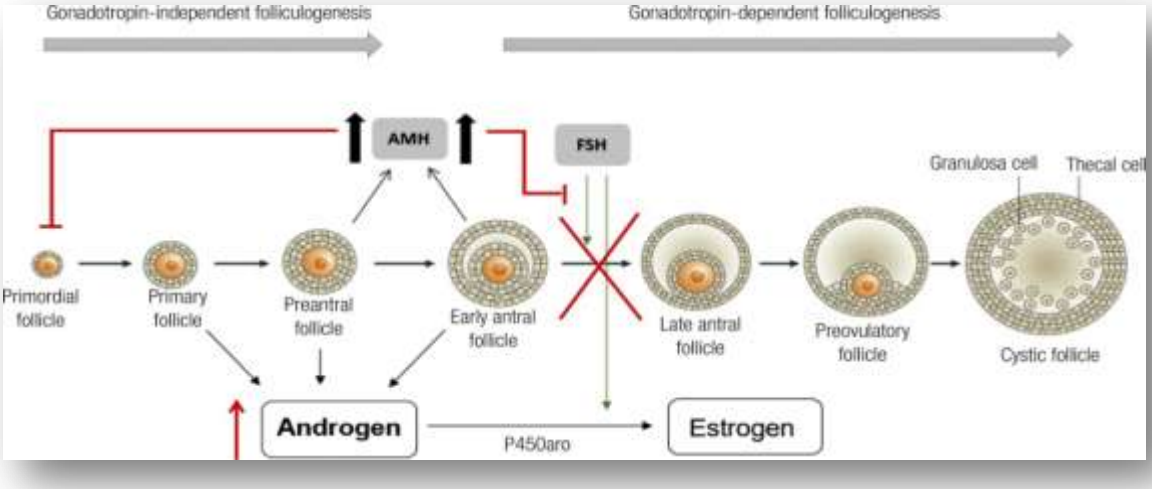

Figure: 2 Role of AMH in folliculogenesis[16].

2. HYPERINSULINEMIA LEADS TO HYPERANDROGENICITY[17,18]

Hyperinsulinemia leads to decline in hepatic synthesis of SHBG and IGFBP1.Decline in the level of SHBG will lead to excess bioavailabity of free androgens,while decline in IGFBP-1 will help in increasing level of circulating IGF-1.IGF-1 and insulin are structurally and chemically similar and the receptors of IGF-1 are theca cells of ovary. Elevated levels of insulin through

IGF-1 receptors will amplify LH mediated thecal androgen production.Hence the ultimate consequence of hyperinsulinemia is hyperandrogenism.

3. OBESITY AND PCOD

Central obesity is common and body fat distribution (waist: hip ratio)appears to be more important in the pathogenesis of PCOD. Obesity augment the metabolic disorder leading to severe menstrual disturbance, oligo-amenorrhea, chronic anovulation, lower pregnancy rates,higher miscarriages rates and increase obstetric complications.[19]Obesity is associated with insulin resistance and hyperinsulinemia which have been linked with hyperandrogenemia.

CONCLUSION:

PCOD is a common and heterogeneous disorder of women of reproductive age. The problem in understanding the etiology has been the wide variability in clinical manifestations and no single etiology can explain all spectrum of PCOD.

In *Ayurveda* the main *Nidana* of PCOD are *Vata-Kapha Prakopaka*.These all *Nidana* are due to irregular dietary and lifestyle habits. In present study of 30 patients most common*Aharaja Nidana* were irregular dietary habit, intake of *Guruguna Pradhana Ahara,Atimadhura Ahara sevana*, junkfood habit, aerated aerated cold drinks, *Ajeernashana*, *Anashana* and *Vishamashana*.*Viharaja Nidanas*were *Ratrijagarana*, *Avyayama and Vegadharana* in present study**.***NidanaParivarjana* is necessary to management of PCOD.

In modern, etiological factors of PCOD are as follows -A group of genes are responsible, but how they are integrated still to be explored. Hyperactive gonadotropin leads to LH excess,excess ACTH production and increased activity of intrinsic ovarian and P450C17 are responsible for hyper and

rogenism.Hyperinsulinemia evidently plays a role in the etiopathogenesis of hyperandrogenism by increasing ovarian androgen production and decreasing the SHBG concentration.Obesity increases the risk of the diabetes in PCOD patient.Hyperandrogenism, Hyperinsulinemia and Obesity are strongly implicated in the etiology of PCOD and reduction of these risk factors should be central treatment focus.

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