International Journal of Pharmaceutical Research and Applications
Volume 6, Issue 5 Sep-Oct 2021, pp: 1209-1213 www.ijprajournal.com ISSN: 2249-7781

PCOS: A 20th Century Phenomenon, Aliterature Review

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Date of Submission: 25-10-2021 Date of Acceptance: 03-10-2021

ABSTRACT
Polycystic ovary syndrome (pcos) is a heterogeneous endocrine disorder, leading to several health complications, including menstrual dysfunction, infertility, hirsutism, acne, obesity, and metabolic syndrome. However, its pathophysiology remains largely unknown but many believe that pcos appears to be familial, with its various aspects differentially inherited from one generation to the next. Researchers and clinicians have been trying to understand the complex interaction between genetic and environmental factors involved in the pathogenesis of polycystic ovary syndrome (pcos) for decades without any consensus being achieved. Comprehensive international guidelines have outlined recommendations for the diagnosis and management of pcos but do not address the pathogenesis. Studies shows that an estimated one in five (20%) indian women suffer from pcos. If not monitored in time, the condition can have serious health impacts. Pcos affects between 2 in 10 or 3 in 20 women worldwide. As per the results of a large scale survey conducted across india in 2020, about 16 percent of women respondents between the ages of 20 and 29 years suffered from polycystic ovary syndrome. Polycystic ovary syndrome occurs due to increased incidents of unhealthy eating patterns and irregular exercise.

Keywords : PCOS, Life style changes, Risk factors, Endocrine disturbing factors, Obesity

I. INTRODUCTION

Polycystic ovarian syndrome (pcos) is a condition which leads to growth of ovarian cysts. It is a prevalent endocrine disorder in child-bearing women, which can lead to infertility. A recent study has revealed that about 18% of the women in india, mostly from the east, suffer from this syndrome. Pcos is characterized by weight gain, irregularity in menstrual cycle, oily skin and darkened acne marks, hypertension and metabolic abnormalities/dysfunction. These can lead to dangerous illness like type 2 diabetes-gestational diabetes, cardiovascular and cerebrovascular dysfunction and higher risk of mood and anxiety disorders which can cause depression.[1]

pcos is a syndrome that has emerged to be-come a more frequent event in the 20th century. If this postulate is true, there are major implications for how we investigate the patho-physiological mechanisms and etiology of pcos and its co-morbidities. A better understanding of how environmental factors and genetics interact, and the nature of the critical environmental exposures, is required to develop effective syndrome-specific interventions and treatments.

lifestyle + metformin is associated with lower bmi and subcutaneous adipose tissue and improved menstruation in women with pcos compared with lifestyle ± placebo over 6 months. Metformin alone compared with lifestyle showed similar bmi at 6 months. These results suggest the combination of lifestyle with metformin has a role to play in weight management.[2]

from the study, noted an interesting fact that the prevalence in rural population is less while compared to those from urban area. However the reason for comparatively less pcos cases among rural population may be due to lack of awareness and/or mini mized or nil exposure to junk foods, pollution and other endocrine disruptors. Moreover girls in rural areas do not depend on labour saving devices for household work or vehicles for transport thus helping them maintain a good bmi.

Polycystic ovary syndrome (pcos) appears to be an ancient disorder, which has persisted in human evolution despite reduced fecundity because of the benefits to affected women such as greater sturdiness and improved energy utilization, a rearing advantage for their children and kin, and a reduction in the risk of perinatal mortality. This raises the possibility that gene variants that are

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eventually found to be associated with pcos will be similar across ethnic groups and races.[3]

II. METHODS

Reviewed available evidence and summarized available data sources on: polycystic ovarian disease (pcod), also known as polycystic ovary syndrome (pcos) is a very common condition affecting 5% to 10% of women in the age group 12–45 years. It is a **problem in which a woman's hormones are out of balance**. It can cause problems with menstrual periods and make it difficult for her to conceive. Doctors don't know exactly what causes pcos. Studies concludes that high levels of male hormones prevent the ovaries from producing hormones and making eggs normally. Genes, insulin resistance, and inflammation have all been linked to excess androgen production.[4]

Risk Factors

- Family history of diabetes
- Family history of infertility
- Lack of physical exercise
- Low grade inflammation
- Intake of saturated, hydrogenated fats
- Endocrine disturbing compound (edc)
- Racial factors
- Neuroendocrine factors

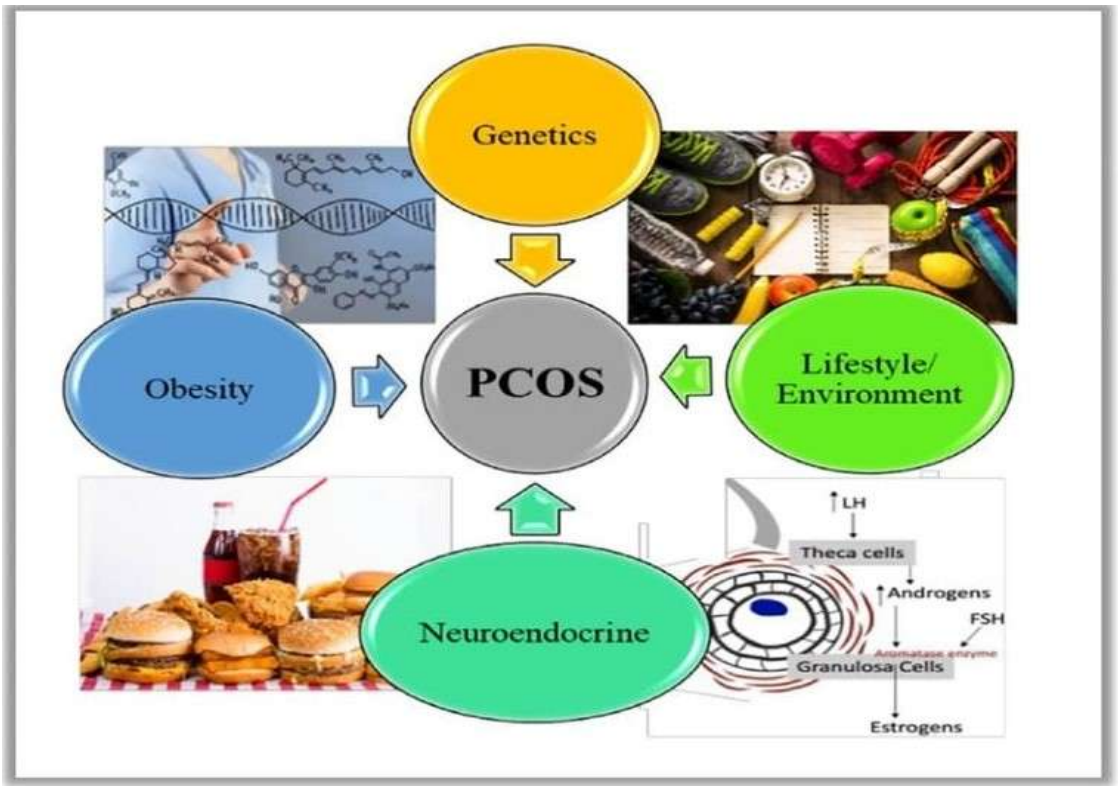

Fig 1. Factors contributing PCOS

Family history of diabetes

Paternal history of diabetes mellitus affects the prevalence and phenotype of pcos. Chen cheng, haolinzhang, [...], and jieqiaoet.al concludes that the paternal or maternal history of diabetes mellitus (dm) contributes to the prevalence and phenotype of polycystic ovary syndrome (pcos).fa [5]

Family history of infertility

The etiology of this syndrome remains unclear but there are strong evidences for a major genetic component in the etiology of pcos. Cases of pcos cluster in families' revealed hereditability of both hyperandrogenaemia and hyperinsulinaemia in affected siblings. Pcos is **known to be inherited genetically with the autosomal dominant**

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manner and 50% of chances are documented of inheritance from mother to daughter.[6]

Lack of physical exercises

Lack of physical activity and excess body weight as potential contributing factors to insulin resistance, according to the national institute of diabetes and digestive and kidney diseases. Obese pcos patients show more difficulty in losing weight by exercise than lean pcos patients. The role of hormonal alterations and pcos per se in the responsiveness of weight loss to exercise remains to be determined.

Long-term maintenance of weight loss among obese population is less likely this issue is exaggerated in obese pcos subjects due to impressive correlation between metabolic characteristics, lifestyle such as physical activity and dietary intake, and obesity.

Although lifestyle modifying measures, such as diet control and exercise, could play an important role in treatment of pcos[7]

Low grade inflammation

It is apparent that pcos is **associated with a significant elevation of multiple markers of inflammation** including crp, il-18, mcp-1, and white blood count. Furthermore, pcos is associated with other derangements associated with inflammation such as increased oxidative stress and endothelial dysfunction. Chronic low-grade inflammation has emerged as a key contributor to the pathogenesis of polycystic ovary syndrome (pcos). A dietary trigger such as glucose is capable of inciting oxidative stress and an inflammatory response from mononuclear cells (mnc) of women with pcos, and this phenomenon is independent of obesity.

Intake of saturated, hydrogenated fats

Foods which have saturated or hydrogenated fats in, include dairy products such as cream or cheese and fatty red meats, as well as processed or fried foods. These unhealthy fats can increase estrogen production, which can make your pcos symptoms worse, and can lead to weight gain which can also worsen symptoms. Foods to avoid in general, people on a pcos diet should avoid foods already widely seen as unhealthful.

These include: refined carbohydrates, such as mass-produced pastries and white bread. Fried foods, such as fast food. Sugary beverages, such as sodas and energy drinks. Processed meats, such as hot dogs, sausages, and luncheon meats. Solid fats, including margarine, shortening, and lard. Excess red meat, such as steaks, hamburgers, and pork. [8]

Endocrine disturbing compounds

The epigenome undergoes significant remodeling during tissue and organ development, which coincides with a period of exquisite sensitivity to environmental exposures. In the case of endocrine-disrupting compounds (edcs), exposures can reprogram the epigenome of developing tissues to increase susceptibility to diseases later in life, a process termed "developmental reprogramming." Both dna methylation and histone modifications have been shown to be vulnerable to disruption by edc exposures, and several mechanisms have been identified by which edcs can reprogram the epigenome. These include altered methyl donor availability, loss of imprinting control, changes in dioxygenase activity, altered expression of noncoding rnas, and activation of cell signaling pathways that can phosphorylate, and alter the activity of, histone methyltransferases. This altered epigenomic programming can persist across the life course, and in some instances generations, to alter gene expression in ways that correlate with increased disease susceptibility. Together, these studies on developmental reprogramming of the epigenome by edcs are providing new insights into epigenomic plasticity that is vulnerable to disruption by environmental exposures. [9]

Example of common edc sources:

- Industrial chemicals and pesticides can leach into soil and groundwater, and make their way into the food chain by building up in fish, animals, and people.
- Non-organic produce can have pesticide residues
- Some consumer products contain edcs or are packaged in containers which can leach edcs, such as household chemicals, fabrics treated with flame retardants, cosmetics, lotions, products with fragrance, and anti-bacterial soaps
- Processed foods can accumulate traces of edcs that leach out of materials used in manufacturing, processing, transportation, and storage
- Soy-based products contain phytoestrogens, which are chemicals produced by plants that mimic estrogen
- Household dust can contain edcs such as lead, flame retardants, and pcbs from weathering construction material or furniture

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Racial factors

The differences in phenotype and clinical symptoms of pcos related to the clinical, hormonal, and metabolic characteristics among various ethnic backgrounds, including hispanics, africanamericans, asians, and indians, need to be considered when assessing and treating these individuals. Particularly, women of different ethnicities had different presentations of clinical hyperandrogenism such as hirsutism which strongly suggests that clinical hyperandrogenism related history taking and physical examination should vary from patient to patient according to different ethnicities.

It found that caucasian females living in the us and europe are less likely to develop pcos compared with females residing in the middle east whereas black women (the majority are african-americans and afro-brazilians) tend to have the highest risks of developing pcos. The upper bound of the 95% cri of the pcos prevalence for white females is the same as the lower bound of that for black females, suggesting that white and black females have substantially different risks of developing pcos.chinese women were suggested to be at a lower risk of pcos compared with other ethnic groups. It should be noted that for females residing in the middle east, in general, we would expect that under the same diagnostic criterion of pcos, chinese women are at a lowest risk of developing pcos, and then in an ascending order through caucasian women and females residing in the middle east, with black women having the highest risks of developing this syndrome. The genetic ancestry data may be used to interpret the phenotypic variability associated with pcos to a greater extent than self-reported ethnicity. There are evidences for genetic influence based on european ethnicity in women with pcos and a genetic component in the phenotypic features of pcos within a mixed european population.

Neuroendocrine factors

Increased lh pulse frequency, lh pulse amplitude, and increased lh/fsh ratios are described in women with pcos. The initial features of pcos emerge during the early pubertal years, concomitant with reactivation of the hypothalamic gnrh pulse generator, increased gonadotropin secretion, and subsequent increased ovarian estrogen production. Loci identified in the genome-wide association studies (gwass) studies include lhcgr, fshr, and fsh-β polypeptide (fshb) genes, emphasizing neuroendocrine contributions to pcos pathophysiology.

III. CONCLUSION

From the study, it is noted an interesting fact that the prevalence in rural population is less while compared to those from urban area. However the reason for comparatively less pcos cases among rural population may be due to lack of awareness and/or minimized or nil exposure to junk foods, pollution and other endocrine disruptors. Moreover girls in rural areas do not depend on labour saving devices for household work or vehicles for transport thus helping them maintain a good bmi. We may note that the number of obese women among the urban population is higher than those among the rural population. Also, family history is found to have the strongest association with the disorder with a high significance. Thus more genetic studies are required to unravel the genetic pathology of this multi faceted syndrome. Also, family history is found to have the strongest association with the disorder with a high significance. Thus more genetic studies are required to unravel the genetic pathology of this multi-faceted syndrome. Pcos may arise from a combination of genetic predisposition and environmental insults that lead to failure of reproductive and metabolic functions. Environmental exposure may start in utero, persist postnatally until adolescence, when pcos becomes clinically evident, and extend throughout life.

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