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POLYCYSTIC OVARY SYNDROME-A REVIEW ON EPIDEMIOLOGY, PATHOPHYSIOLOGY & HERBAL TREATMENT

Vivek kumar[1], Palak Panjwal[1],Kanika Sharma[1], Pravesh Kumar[1],Swati Kashyap[1], Neelam Sharma[1]*

[1]Himachal Institute Of P'ceutical Education & Research, Nadaun, HP

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Abstract:

The heterogeneous illness known as polycystic ovarian syndrome (PCOS) affects females and is connected to an endocrine reproductive disorder. It affects girls of 18-44 age. Complexities like many cysts and an irregular menstrual cycle are caused by the ongoing hormonal imbalance, which eventually makes it difficult for women to conceive. Several potential genes have been shown to be one of the factors contributing to PCOS. To determine the genetic linkage of PCOS, numerous investigations have been conducted. It is crucial to conduct research that pinpoint the hormone imbalance, and PCOS's precise etiology. This review has emphasized on epidemiology, genetics, diagnosis and treatment of PCOS, which causes hormonal imbalance. Yet, this study was only a superficial attempt to understand the pathophysiology and genetic basis of PCOS.

Keywords: *PCOS, AMH, hyperandrogenism, oligomenorrhea, Infertility.*

Corresponding author:

Neelam Sharma,

Department of Pharmacology

Himachal Institute of Pharmaceutical Education &Research, Nadaun.

Email id:bhardwajneelam1992@gmail.com

QR code

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INTRODUCTION:

Polycystic ovarian syndrome (PCOS) is a hormonal imbalance. Unlike other ovulation diseases in which the ovaries are non-functional or defective, this syndrome is distinguished by persistent anovulation and ovarian dysfunction. Nowadays, the majority of therapy is focused on the patient's primary complaint. The goal of treatment is to reduce hyperandrogenism symptoms, restore menstrual regularity, and achieve pregnancy.[1]

PCOS appears to be a complicated characteristic caused by the combination of several hereditary and environmental variables. PCOM, hyperandrogenemia, insulin resistance, and insulin secretory abnormalities are all heritable.[2]

PCOS is a genetically complex endocrine condition characterised by hyperandrogenism (acne & hirsutism) and anovulation (infertility, oligomenorrhea, and dysfunctional uterine haemorrhage) in women with polycystic ovaries. In many countries, it is the major cause of female infertility.[3]

In 5-20% of reproductive aged women worldwide, PCOS is a hormonal and metabolic disorder that causes androgen excess, menstrual dysfunction and oligo- ovulatory subfertility.[4]

PCOS is linked to a bigger hazard of metabolic abnormalities such as insulin resistance and hyperinsulinism, type 2 diabetes, dyslipidemia, cardiovascular disease, and endometrial endometrial, cardiovascular disease, dyslipidemia, type 2 diabetes, and insulin resistance and hyperinsulinism cancer. [5-8]

It has been postulated that instability in the neuroendocrine system leads to an imbalance in the hypothalamic-pituitary-ovarian axis, resulting in gonadotrophin overproduction in women with PCOS. An increase in hypothalamic GnRH promotes the production of the -subunit of LH over the development of the -subunit of FSH, which favours the production of LH over FSH.[9-10]

A single follicle grows and experiences ovulation in a typical fertile female from a pool of primordial follicles present in the ovaries since birth. The pace at which primordial follicles are chosen for development is closely regulated in order to preserve ovarian reserve and fertility. An imbalance between androgens, anti-Müllerian hormone (AMH), and follicle stimulating hormone (FSH), causes follicular growth to stop in PCOS. AMH is generated by granulosa cells in the ovary and is vital in preventing primordial follicles from developing into primary follicles. The polycystic appearance of the ovaries in PCOS is caused by a significant number of primordial follicles developing and then stopping.[11-13]

These women have a higher hazard of pre-eclampsia, foetal macrosomia, gestational diabetes, small-for-gestational-age children besides postnatal mortality if they become pregnant .[14-16]

The most typical gynecologic symptom of PCOS is irregular menstruation. 85 to 90 percent of women with PCOS experience oligomenorrhea, and 30 to 40 percent of patients who experience amenorrheas do as well. [17]

Stein-Leventhal syndrome and hyperandrogenic anovulation are other names for it (HA). It is also known as syndrome "O," which stands for excessive nutrient intake, excessive insulin production, and ovary disturbance. It is connected to the type-2 diabetes development such as recurrent miscarriages. [18]

The PCOS patient ovaries includes higher than 10 follicles seen on ultrasound. The polycystic ovary, in compare with normal ovary, its layer has higher follicles and have thick centre. This centre is called stroma which is where testosterone made. [19]

Normally, PCOS includes the presence of any three criteria, clinical or biochemical evidence of hyperandrogenism, oligoovulation and the presence of polycystic ovaries on ultrasound examination. However, ovaries are non- specific finding noted in women with no endocrine or metabolic abnormalities.[20]

Computed tomography scans, Ultrasound, magnetic resonance imaging (MRI) are frequently used to detect PCOS (CT). [21]

Stein and leventhal first identified PCOS in a group of seven patients who had polycystic ovaries, amenorrhea, infertility and hirsutism in 1935.(Stein and Leventhal, 1935).The endocrine disruption in women is mostly caused by ovarian hyperthecosisand increased androgen production in PCOS. Various genetic and environmental variables are also present and have a part in the pathophysiology of this condition.[22]

PCOS has negative neurological and psychological effects on one's quality of life, including a depression, in addition to cardiovascular problems, endometrial and breast cancer, and other conditions. Up to 20% of women with reproductive issues (including fecundity and early miscarriage) have been given a PCOS diagnosis.[23]

Based on US data and projections of Australia's historically lower frequency, the expected economic cost of PCOS in 2006 was $400 million (infertility and 31% menstrual dysfunction 12% and diabetes linked to PCOS accounts for 40% of total expenditures) posing a significant health and financial burden.[24]

Gonadotropines are frequently used to induce ovulation in clomiphene-resistant women, however this procedure needs to be closely watched reduce the danger of ovarian hyperstimulation. There have been several dosing dosing regiments utilized, with the low dose step-up regimen being the most effective and secure for use. [25]

The prevalence of PCOS has increased due to the use of many diagnostic criteria, and most recently, it was determined to be 18% (17.8).In the community-based prevalence investigation, 2.8% based on current diagnostic standards from Rotterdam. Importantly this recent study found that 70% of women without ultrasound, estimations of polycystic ovaries (PCO) were used to impute this study. Non-imputed prevalences were determined to be 11.9 – 2.4%.[26]

A hormonal imbalance in women leads to PCOS , a medical disorder. Specifically, higher than normal amounts of LH, androstenddione, DHEA-S, testosterone, or low level of oestrogen. Insulin resistence, high blood sugar, and in women with PCOS, poor glucose tolerance is fairly common. Yet slim women with PCOS may also experience insulinresistence.[27]

PCOS patients are frequently obese, but the prevalence of overweight and obesity varies from series to series, in part due to type of clinic the patient is initially referred .Hence the percentage of overweight/obese people our own reproductive endocrinology clinic's patient (35-40%) is considerably less than that indicated that indicated in the clinic in which metabolic medicine is the primary focus (usually 480%).[28-29]

PCOS's precise cause is still a mystery. New research indicates that inflammation may be one of the possible risk factors for speculate that the derogatory Chomkines and cytokines were regarded as two of the hall marks in PCOS that activate the immune system. [30]

PCOS is frustrating experience for women, frequently challenging for treating professionals , and a difficult scientific problem for researchers.[31]

A polycystic ovary shape affects about 20% of women who consider themselves to be reproductively healthy (10). Despite having regular, ovulatory menstrual cycles, many of these women display the biochemical symptoms of the condition , including high levels of testosterone and/or luteinizing hormone (LH). Even so, some have ovulatory menstrual periods and normal amounts of reproductive hormones. [32]

The main categories of ovulations-including therapies for PCOS patients are those that directly target the hypothalamic-pituitary-ovarian axis, including those that effect metabolic variables, which most likely cause indirect disruption of the hypothalamic-pituitary-ovarian axis. [33]

Women with a hypothalamic pituitary ovarian axis is a painstakingly coordinated with strictly controlled webbing that is ultimate in charge of the species ability to reproduce and survive. The women hypothalamus pituitary ovarian axis is a painstakingly orchestrated and rigidly regulated network that ultimately control capacity of species to procreate and thrive. Both internal cues, such as neuronal as well as hormonal ones, external inputs cause the hypothalamus. Pituitary ovarian axis to response. The factors being to have an impact throughout pregnancy and spread to the developed in brain and germ cells. The production of androgen has been increased and observed in studies using ovarian theca cells from PCOS patients and increased the result of ovarian steroidogenesis, which is primarily attributed to altered expression of key enzymes cytochrome P450 enzymes: CYP19,CYP21, CYP11A and CYP19 in the steroid hormone biosynthesis pathway.[34-35]

Impaired insulin sensitivity and adult onset diabetes, cardiac arrest issues, neurological or psychological side effects (like apprehension and misery) and uterine cancer are all connected with PCOS. PCOS has been determined in many 20% females with sterile issues.[36]

EPIDEMIOLOGY:

As per frequency estimates polycystic ovarian syndrome is a recurrent dysfunction of an endocrine

gland that influences 4 to 8% females of fertile age as per to the NIH/NICHD criteria.[37]

Polycystic ovary syndrome was detected in Whites and Blacks using the 4.0% NIH 1990 criteria. The next study included 400 randomly selected consecutive females between the ages of 18 and 45 in the prevalence of PCOS in the same setting was 6.6%, yet there was still no noticeable difference between Black and White. (8.0 and4.8%, respectively).[38]

The issue is whether obesity alone may cause PCOS. Obesity is linked to low levels. Higher levels of free testosterone induced by SHBG and delayed follicular phases (without anovulation) result in a lengthier menstrual cycle, which may be misinterpreted with a PCOS diagnosis.[39]

Although one would expect a higher prevalence than the 6.5-8.0%, the prevalence of PCOS diagnosed by more comprehensive standards, such as the Rotterdan 2003 criteria, is less obvious.[40]

Estimated lifetime risks for women with mutations in DNA repair genes such as BRIPI, RAD51C, and RAD51D are 5.8%, 5.2%, and 12%, respectively.[41]

North America and Central and Eastern Europe have the greatest age-adjusted incidence rates, generally exceeding 8 per 100,000 people. Asia and Africa have the lowest rates (3 per 100,000), whereas South America has an intermediate rate (5.8 per 100,000). Migration from countries with low rates to those with high rates increases the danger.[42]

Because there are 62 million women in the United States between the ages of 15 and 44, we may infer that at least 4-5 million of them are of reproductive age.[43]

Insulin resistance was found in 75% of lean PCOS patients, 62% of overweight controls, and 95% of overweight PCOS patients using a hyperinsulinemic euglycemic clamp test.[44]

The prevalence of PCOS in those who are underweight, normal weight, overweight, and mildly, moderately, or severely obese. Women made up 8.2%, 9.8%, 9.9%, 5.2%, 12.4%, and 11.5% of the population, respectively. The authors came to the conclusion that obesity may be on the rise. The potential of PCOS, however the impact was limited. Women with epilepsy have a greater frequency of reproductive illnesses such as PCOS.[45]

We evaluated nearly 300 PCOS patients who visited a single reproductive endocrine clinic and observed that hirsutism and ovulatory impairment were much worse in fat women than in lean females.[46]

PATHOPHYSIOLOGY:

PCOS is likely a heterogeneous condition, with different disturbance leading to a variety of ultimate phenotypes. A significant advancement in our understanding of the pathophysiology of PCOS would be the discovery of a mechanism that combines hyperandrogenism, inadequate insulin sensitivity, and follicular arrest into an unified phenotype.[47]

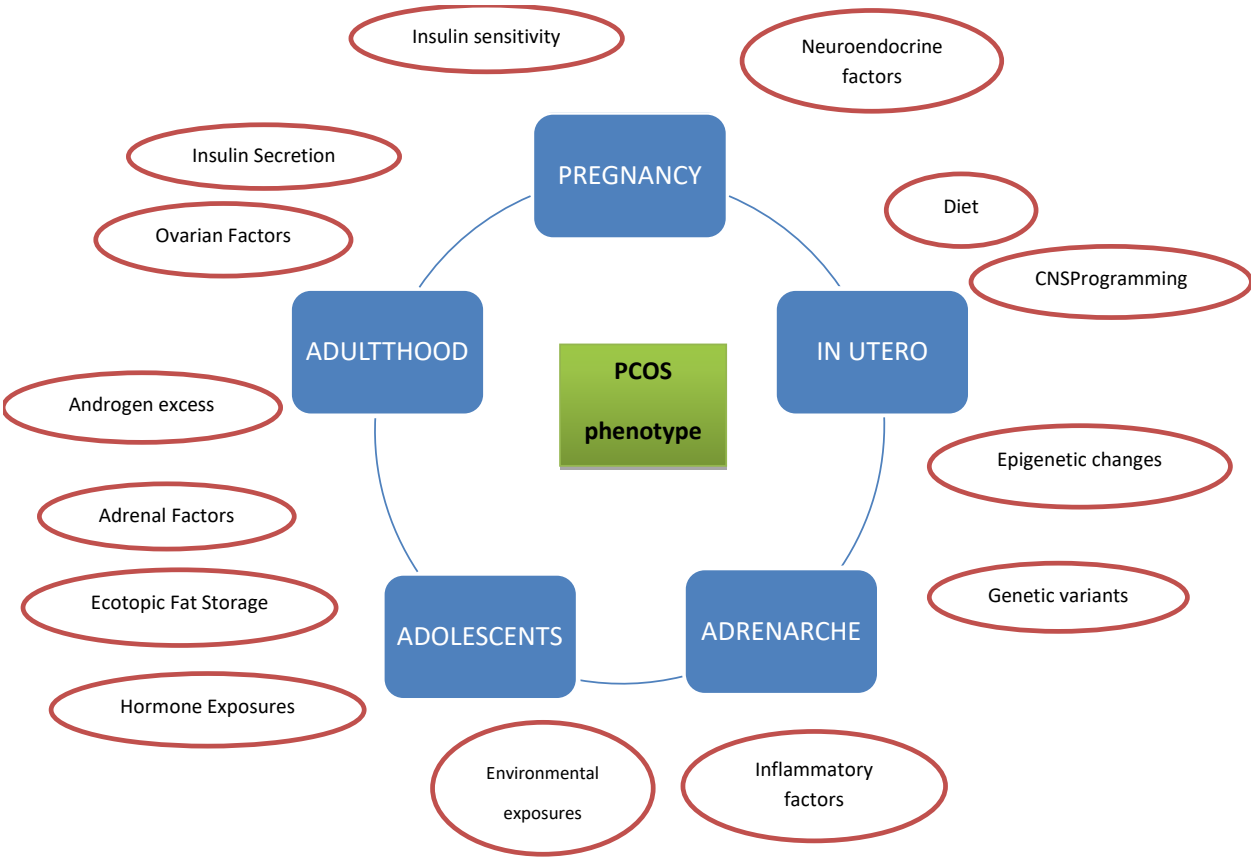

FIGURE 1: Numerous theories have been build over the years on primary anatomical factor of Polycystic ovarian syndrome . Polycystic ovarian syndrome is a result of interactions between numerous proteins and genes that are altered by environmental and epigenetic factors[48]

Insulin resistance is a significant component of this condition. Regardless of weight, the majority of female with polycystic ovarian syndrome with undiagnosed kind of intrinsic insulin impaired insulin sensitivity.[49-50]

BahriKhomami, Mahnaz, et al. "Increased maternal pregnancy complications in polycystic ovary syndrome appear to be independent of obesity—A systematic review, meta-analysis, and meta-regression." Obesity Reviews 20.5 (2019): 659-674.[51-52]

Anti-mullerian hormone (AMH), a crucial predictor of ovarian reserve, and vascular endothelial growth factor (VEGF), a critical component of angiogenesis, have lately come under scrutiny as probable culprits in the development of PCOS in people of all ages. AMH levels have been linked to circulating androgens, insulin resistance, and an AMH function. PCOS has a suggested pathogenesis.[53]

In terms of gonadotropins, women with polycystic ovary syndrome often have greater circulating LH concentrations and FSH levels that are low to normal. When patients with polycystic ovarian syndrome are compared to women with normal menstrual cycles, the frequency of LH pulses (as a reflection of gonadotropin-releasing hormone pulsatility) is usually increased.[54]

As a result of having a higher waist-to-hip ratio, or centripetal fat distribution, women with PCOS are more prone to dyslipidemia. The central adipocytes appear to negatively affect blood lipid levels.[55]

PCOS may not just be a female reproductive condition. We can deduce that it is more likely a

metabolic illness (with reproductive failure) that affects both sexes. The two reproductive symptoms of PCOS that are only present in women are Polycystic ovaries and anvulatory diseases.[56]

Adrenal hyperandrogenism, which is typical of PCOS patients, is dependent on androgen production rather than hypothalamus-pituitary adrenal gland axes mediated response.[57]

A powerful metabolic marker of coronary heart disease, HDL cholesterol, is considerably reduced in women HDL-cholesterol levels that were greater than usual in despite the fact that difference was not substantial, PCOS subjects after correcting for additional factors like body mass index (BMI) and insulin fasting. [58]

PCOS is most likely a diverse condition with different agitation leading to a variety of ultimate morphologies. We have made significant progress in our understanding of the pathophysiology of PCOS with the discovery of a pathway connecting hyperandrogenism, decreased sensitivity, and follicular arrest in one phenotype. [59]

This hyperandrogenism is brought on by increased P450c17 enzyme concentration. According to a study (Draper et al.,2004), PCOS hyperandrogenism is influenced by genetic and epigenetic variance. A lack of cortisone reductase is an illustration of a hereditary aetiology of PCOS with adrenals. Hydroxysteroid dehydrogenase 11 Cortisone is not converted to cortisol in type 1 deficiency, which raises the ACTH level even more level and causes the synthesis of excess androgens.[60]

Symptoms:

Later, in twenties or thirties, symptoms such as the onset of first period or potential health problem began. These signs can vary from woman to woman but has yet to receive a polycystic ovarian diagnosis. Polycystic ovarian syndrome one requires at least twoof these problems.

Puberty unrelated acne, Fatigue, Excessive growth of hair, Absence of periods, Skin turning darker, Retention of fluid, Infertility, Mood swings, Gaining weight, Extended or intense periods, Irregular menstrual cycles, Obstetrical cysts, Period discomfort, Baldness on the male pattern, Hirsutism, Anovulatory infertility. [61-62]

Genetics:

According to twin studies, hereditability is 70%. Only a small percentage of the predicated hereditability can be explained by the few genetics loci that have been found. At least 16 PCOS susceptibility loci have been found in GWASs of Han Chinese and European women. [63-64]

The genetic basis of PCOS varies between and within families, yet it is connected to a common pathway Due to complexity and variability, a single and connected gene. No chromosomes from one family is recorded. Different chromosome have varying hereditaey predisposition in patients of similar family .[65]

A complicated interaction of environmental factors, genetic vulnerability, and protective genetic variations affects PCOS, a complex polygenic condition. In the past, studies on the genetics of PCOS have revealed that the condition is inherited in an autosomal dominant manner.[66]

The role of heredity in this complex condition has been investigated using techniques like twin studies and linkage studies. Linkage studies follistatin, one of 37 potential genes, was expected to have high correlation with infection, and the YP11A1 gene, a nominal association. Siblings with PCOS-related features and hyperandrogenismThe transmission disequilibrium test revealed a strong genetic relationship of D19S884 allelic marker near the INSR gene related with PCOS in this research.[66-67]

Female Androgen Receptor gene (AR). This gene, which is found on chromosome xq12, codes for a protein that is over 90 kb long and has a total of three functional domains. It has 11 exons.[68]

The field of PCOS research known as genome-wide association studies (GWAS) Has emerged as a promising one. Since their debut in 2005, GWAS have been used to scan complete genomes and identify susceptibility loci in a variety of disorder, such as type 2 diabetes, Crohn's disease and asthma. Only few GWAShave been conducted so far on PCOS (21,82). The initial GWAS included a set of 744 PCOS – afflicted women and 895 Han Chinese controls. 5012 controls and 2840 PCOS patients were included in the two replication cohorts and 498 PCOS patients and 780 healthy women, respectively.[69-70]

There are a few genes and multiple loci for which there is strong evidence supporting their involvement in which could leads to a consensus in the soon after. Among these loci is CYP110 (five studies), the insulin

gene's VNTR (six CAPN10 (three trials), D19S884 (three studies), and (Two studies).[71-72]

Although many genes have been researched as potential susceptibility loci, the impact of anyone gene may be minimal ifPCOS is in fact a complex genetic illness. Because of this an the issue of ethnic diversity in many populations, conclusive case-control (or family-based) large number of cases and controls are necessary for investigations (at least 300 per group),homogenous population, and replication in other, independent populations all of which are crucial.[73-77]

Aromatase is one of the complicated Cytochrome P450 family steroidogenesis enzymes and generally plays a key part in steroidconversion. It facilitates the transformation of testosterone into oestrogen. A flaw in the route caused by a lack of aromataseprevents its conversion.[78]

Diagnosis:

For PCOS diagnosis, women must satisfy two of three criteria: polycystic ovary morphology on ultrasound with exclusion of other disease, clinical and/or biochemical hyperandrogenism, oligo-ovulation or anovulation.

Ultrasounds show PCOS in 25% of young women overall, and its inclusion in diagnostic standards has boosted the occurrence of PCOS. According to recent evidence, using the ESHRE may treble the prevalence of PCOS. ASRM criteria with 12% prevalence (not attributing) between 18% (imputing presence of polycystic ovaries) and presence reported in a community sample with polycystic ovaries.[80]

In follicular fibroblasts produced from PCOS women, Yang et al. found that there was less aromatase expression and more testosterone.[81]

Other illnesses linked to irregular menses and/or hyperandrogenism must be ruled out before adult women are evaluated. These conditions include Cushing syndrome, 21-hydroxylase deficiency that is typically nonclassical, androgen-secreting, tumours, hyperprolactinemia, thyroid dysfunction, and CAH. They can also include exterior apply of corticosteroid and androgenic hormone.[82]

An investigation was carried out in 2006 by task force established by the Androgen Excess & PCOS Society (AEPCOS), which was made of five US researchers and six from Europe and Australia. Comprehensive evaluation of the existing literature to establish the connection between independent morbidity and PCOS characteristics. They determined that PCOS is mostly a condition of Androgen overproduction and that a quick PCOS diagnosis depending on existence of clinical or biochemical. Ovarian dysfunction in conjunction with HA (OD or PCOM),eliminating other factors.[83-84]

Among women with PCOS, infertility affects 40% of them. The most frequent reason for anovulatory infertility is PCOS. Infertility clinics see patients with PCOS. There are noticeably more primordial follicles now. However, once follicles are 4 to 8 mm in diameter anomalies in the chemicals necessary for healthy follicular development cause follicular growth to stop. Ovulation does not take place because a dominant follicle does not form.[85-86]

After ruling out other conditions linked to irregular menstruation or hyperandrogenism, adolescent girls with persistent oligomenorrhea for 3 - 4 years postmenarche and clinical and biochemical hyperandrogenism can be diagnosed with PCOS. These patients can be deemed the chances of PCOS if their Oligomenorrhea has not continued for 2 years and need regular evaluation to detect any recurrence. PCOS characteristics referred diagnosis makes an effort to prevent overdiagnosis, which has the potential for early categorization, worry and pointless interventions. Patient's demand for identification and a specific treatment approach must be balanced with diagnostic labeling, though interventions.[87-90]

There are several useful uses for the phenotypic approach to PCOS definition. For instance, it would be beneficial to detect women with infertility in routine clinics practice. Patients with PCOS who are most at risk for metabolic dysfunction people who have "classic" PCOS traits, or phenotypes A and B. [91]

When performing clinical trials and epidemiologic research, another significant use of this strategy is shown where the usage of this classification enables researchers to classify their findings based on a limited range of PCOS characteristics, allowing comparisons with other well defined populations with PCOS.[92]

Treatment:

In general, medical therapy for hirsutism, acne in polycystic ovarian syndrome seeks to lower androgen levels, attenuate there effects by reducing androgen production, increase binding to particular plasma binding proteins and inhibit androgen activity at the level of the target tissue.[93]

In evidence-based approach, changing once life styles is primary line of treatment for the majority of the PCOS women who are overweight. Additionally, it's important to emphasise the prevention of excessive weight gain in all females with PCOS, whether there bodies are regular or large weight. Evan a 5% -10% weight decrease might have a big impact clinical advantages enhancing psychological effects.[94-95]

Long-lasting, more frequent and more intense physical activity improve health maintenance. Importantly, children and adolescents who engage in average to energetic physical exercise atatleast 60 minutes every day have improved physical and mental health. Atleast three times a week, 60 minutes of medium to strenuous physical exercise advised foe PCOS patient to prevent weight gain and maintain their health. Cardiometabolic risk factors can be improved with exercise therapy in PCOS women.[96-98]

Along with patients complaints of irregular mensturation, the PCOS related frequently chronic anovulation can raise a patients risk of endometrial hyperplasia and cancer. Endometrial proliferation be reduced by using cyclic progestin or small amount of dosage CHC combining oesteogen as well as progestin. The main suggested therapy for menstrual problems associated with PCOS and increased menstrual regularity is low dosage CHCs.[99-100]

Metformin can help women with very high blood sugar levels increase their menstrual cycle, ovulation rate or pregnancy rate BMI or being unable to shed pounds. In, 2001 metformin is administered to PCOS affected women as demonstrated by vandermolen et.al. that clomiphene citrate- resistant individuals can raise the ovulation and pregnancy rates in contrast to the oestrogen antagonist.[101]

Type2 diabetes and IR are two metabolic symptoms of PCOS that are improved by metformin . The elevated risk factors for various related health issue like obesity and cancer are greatly influenced by these phenotypes .In recent years, the usage of insulin–sensitive medications similar to metformin has been demonstrated to not only relieve metabolic symptoms but also result in cancer incidence is declining. Anovulation, a typical PCOS characteristic, has also been demonstrated endometrial cancer.[102]

The antiestrogen clomiphene citrate is first medication of choice for female with newly diagnosed polycystic ovarian syndrome. It stimulates pituitary gonadotropin release, causing follicular recruitment as a result. The majority of PCOS patients will ovulate with the medicines. The majority of PCOS patients will ovulate with the medicines. This medication is simple to administer, risk-free, offordable and efficient.[108]

With long-term survival rates, bariatric surgery has gained popularity as a way for obese men and women to lose weight and keep it off (160). There has recently been interest in the usage of bariatric surgery as a treatment for PCOS- afflicted women who are excessively obese. Escobar and colleagues conducted research. The effects of weight loss following bariatric surgery on PCOS traits.[109]

Use of herbal in management of pcos:

Aloe Vera: Traditional medicine has employed Aloe Barbadensis Miller to treat condtions like arthritis, burns, skin cancer, digestive issues, diabetes and high blood pressure. Radhaet examined the herb Aloe vera's potential as a preconception aid for treating rats with PCOS.[103]

Atractylodes:

Zhou et al evaluated the effects of a polar extract of Atractylodesmacrocephalakoids(AMK) in a testosterone-induced hyperandrogenic rat model of PCOS propionate. AMK is a tonic plant is frequently utlilised in Chinese medicine for PCOS treatment. 5 grouping of PCOS was induced with testosterone using animals propionate. [104]

Guggul:

Effects of commiphorawightii on rats with polycystic ovarian syndrome(PCOS) were examined by Kavitha et al. Four group of animals were used in the experiment. PCOS was brought on utilizing dehydroepiandrosterone(DHEA). The creatures received Commiphorawightiiresin ethanolic extract I combination with metformin together with DHEA. Glucose levels in the blood and steroid the hormones were counted.[105]

Hazelult:

Demirel et al. also investigated the effects of Corylusavellana seed oil, usually reffered to as hazelnut oil, on the polycystic ovarian syndrome (PCOS) brought on by letrozole. 106]

Turmeric:

Curcuma longa rhizomes contain curcumin. It has anti-inflammatory, antioxidant, antihyperlipidemic, and hypoglycemic characteristics and it is used as a food ingredient. Reddy and evaluation of Curcumin's advantages in female Wister rats using PCOS. There

were five different animal groups. Letrozole was employed to trigger PCOS.[107]

CONCLUSION:

PCOS is the most common metabolic and hormonal imbalance disorder affecting women. PCOS,its epidemiology, pathophysiology, various symptoms, genetics, diagnostic criteria, its treatment and various herbal drugs utilized in the management of PCOS have been the main focus of this review article. It is a condition with various reproductive, metabolic, and cardiovascular components that have long-term health effects. A large portion of the clinical presentation is driven by androgen excess and insulin resistance, both of which have significant hereditary components. The development of macrovascular disease may be aided by the insulin resistance associated with polycystic ovarian syndrome, which appears to raise the risk of glucose intolerance, diabetes, and lipid abnormalities. It is a condition with various reproductive, metabolic, and cardiovascular components that have long-term health effects. A large portion of the clinical presentation is driven by androgen excess and insulin resistance, both of which have strong hereditary components. The polycystic ovarian syndrome's insulin resistance appears to raise the risk of glucose intolerance, diabetes, and lipid abnormalities and may hasten the development of these conditions.

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