IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

"A Review On Pcos Disease In Female Reproductive System"

[1]Chetana.P. Bokriya , [2]Tejashri B. Kadu, [3]Sachin J. Dighade

[1]Student, [2]Asssistant Professor, [3]Principal

[1]Department of Quality Assurance,

[1]Institute of Pharmacy and Research, Badnera, Amravati, India.

Abstract: Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine disorder affecting 6-20% of women of reproductive age, characterized by hyperandrogenism, oligo-anovulation, and polycystic ovarian morphology. This condition is associated with significant health risks, including insulin resistance, obesity, type 2 diabetes, and cardiovascular disease. The diagnosis of PCOS typically follows the Rotterdam criteria, requiring two of three features: oligo-anovulation, clinical or biochemical hyperandrogenism, and polycystic ovaries on ultrasound. The pathophysiology of PCOS involves a complex interplay of genetic, hormonal, and environmental factors. Management strategies focus on symptomatic relief and metabolic health, emphasizing lifestyle modifications such as weight management and exercise. Despite the established criteria for diagnosis, many women remain undiagnosed or mismanaged due to the syndrome's heterogeneous presentation. Ongoing research aims to refine diagnostic approaches and treatment options to improve outcomes for those affected by PCOS.

Index Terms **-** Neuropathic pain, Polycystic Ovary Syndrome (PCOS), Endocrine disorder, Reproductive-aged women, Hyperandrogenism, Oligo-anovulation, Clinical hyperandrogenism, Biochemical hyperandrogenism

I. INTRODUCTION

What is PCOS in women?

PCOS (Polycystic ovary syndrome) is a common condition that affects how a woman's ovaries work. It often results in having too much of a male hormone called Androgen. Many small sacs of fluid develop on the ovaries. They may fail to regularly release eggs. [1]

We do not have a clear view of the etiology of PCOS, although both genetic and environmentalinfluences are implicated. Consequently, there is no single diagnostic marker, whether biochemical or genetic, to provide a gold standard for reference. PCOS is a syndrome and no single diagnostic criterion (such as hyperandrogenism or PCO) is sufficient for clinical diagnosis. PCOS also remains a diagnosis of exclusion. A typical aspect of PCO is excessive androgen biosynthesis. In turn, androgen excess was designated as the main reason for the excess of follicles and the promotion of the interna and granulosa cell proliferation. This effect on folliculogenesis is particularly marked on preantral and antral follicles. Which are very rich in androgen receptors. Indeed, androgen receptor gene expression is highest in granulosa cells of small follicles. [2] PCOS includes a wide spectrum of clinical symptoms and signs. Clinically, diagnosing a woman as having PCOS implies an increased risk for infertility, dysfunctional bleeding, endometrial carcinoma, obesity, type 2 diabetes mellitus dyslipidemia, hypertension, and possibly cardiovascular disease. [2, 3]

Figure No:- 1 (Diagrammatic representation of PCOS)

HISTORY OF PCOS:-

Stein and Leventhal are regarded to have been the first investigators of Polycystic ovary syndrome (PCOS), however in 1721 Vallisneri, an Italian Scientist, described married, infertile women with shiny ovaries with a white surface, and the size of pigeon eggs. It was not until the early 1990s at a National Institute of Health (NIH) sponsored conference on PCOSthat formal diagnostic criteria were proposed and afterward largely utilized.

When was PCOS discovered in India?

PCOS was described in early 1935. However, even today there is a general lack of awareness regarding the condition in India and it often remains undetected for years. This health condition is estimated to affect about 10 million women globally.

Who introduced the concept of PCOS in India?

An estimated one in five (20%) Indian women suffer from PCOS.If not monitored in time the condition can have serious health impacts. Gynecologist Dr. Duru Shah who is also the founder of the PCOS Society of India said PCOS is not a Disease, but a condition that can present itself in different ways.

DIFFERENCE BETWEEN PCOS & PCOD

Table No :- 1 (Difference between PCOS &PCOD)

PCOD	PCOS
It is known as Polycystic Ovary Disease.	It is known as Polycystic Ovary Syndrome.
In PCOD the ovaries start releasing immature eggs that lead to hormonal imbalance and swollen ovaries, among other symptoms.	In PCOS endocrine issues cause the ovaries to produce excess androgen, which makes eggs prone to becoming cysts.
There are less number of cysts on ovaries.	There are more than 10 cysts on the ovaries.
It can be reversed.	It is difficult to reverse this condition.
Generally does not affect fertility.	It can lead to fertility issues.

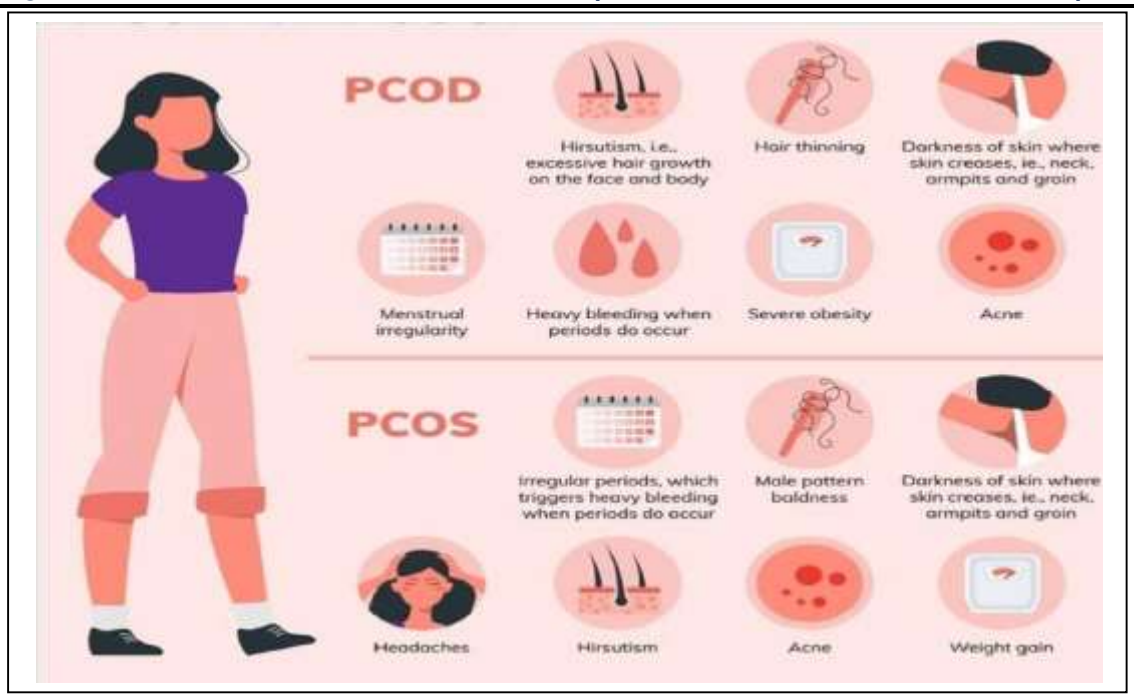


Figure No 2(Understanding your symptoms of PCOD or PCOS)

ETIOLOGY:-

Currently, there is no cause for PCOS. However, there are associations with excess insulin, low-grade inflammation, and genetics. Doctors don't know exactly what causes PCOS. They believethat high levels of male hormones prevent the ovaries from producing hormones and making eggs normal.

GENE:- Studies show that PCOS runs in families. It's likely that many genes not just one contribute to the condition.

INSULIN RESISTANCES:- Up to 70% of women with PCOS have insulin resistance meaning that their cells can't use insulin properly. When cells can't use insulin properly, the body's demand for insulin increases. The pancreas makes more insulin to compensate for extra insulin triggering ovaries to produce more male hormones.

INFLAMMATION: - Women with PCOS often have increased levels of inflammation in their bodies. Being overweight can also contribute to inflammation. Studies have linked excess inflammation to higher androgen levels.

HORMONAL IMBALANCE: - Many women with PCOS are found to have an imbalance in certain hormones, including a raised level of testosterone a hormone, although all women usually produce a small amount of it. [4, 5].

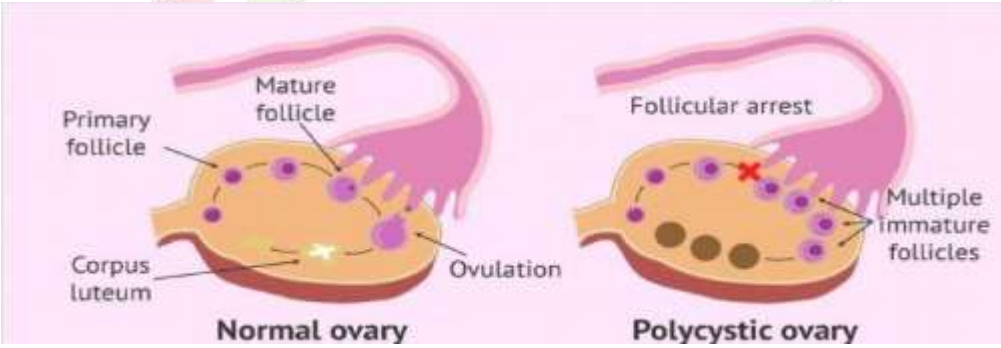


Figure No: - 3 (Differences between normal ovary and polycystic ovary)

SYMPTOMS & SIGNS:-

The onset of symptoms is variable, with some women reporting them in parallel with the onset of menarche, whilst others do not receive a diagnosis until they experience difficulties conceiving. The most common symptoms include:

Irregular menstruation is defined as a menstrual cycle that continuously falls outside the expected cycle length of 24 – 38 days. This may be accompanied by heavy bleeding when menstruation does occur as the uterine lining grows thicker if not shed regularly.

Excess hair growth on the face and body is referred to as hirsutism, caused by excess amounts of androgens. Conversely, hair on the scalp may become thinner and fall out.

Acne typically presents as inflamed cysts around the jawline, cheeks, and chin. This results from an excess of testosterone which causes overactivity of sebaceous glands. Weight gain affects up to 80% of women with PCOS and tends to develop in the abdominal area. [8]

HOW IS PCOS DIAGNOSIS?

Medical history, examination, blood tests, and ultrasounds are used to diagnosePCOS. A PCOS diagnosis requires 2 out of the following 3 criteria.

Table No. 2 (Diagnosis of PCOS)

1) Periods less regular	• More or less often than monthly and No periods
2)Androgens	• Symptoms such as excess hair growthand acne • High levels of male-type hormones(androgens) in the blood
3) Ultrasound	• An ultrasound showing partially developed eggs that look like dark circles (20 or more on either ovary) • Ultrasound is only needed if 1 and2 are not present Not recommended for women agedunder 20.

DIAGNOSIS METHOD OF PCOS

Hyperandrogenism :-

Clinical phenotyping of PCOS involves determining the presence of clinical and biochemical androgen excess (hyperandrogenism) while excluding related disorders. Clinical hyperandrogenism Hirsutism is the best clinical marker of hyperandrogenism, although different degrees of hirsutism should be expected based on ethnicity. Hirsutism is defined as the presence of unwanted terminal (coarse) hairs in females in a pattern more typically seen in adult males. This is in contrast to hypertrichosis, which is independent of androgen influence and is manifested by the superfluous and uniform growth of non-terminal (vellus) hair over the body, particularly in nonsexual areas. Visual assessment of the degree of excess terminal hair can be made using the Ferriman-Gallwey scale. The original scale scored hair density on a scale of 1 at 11 androgen-sensitive sites (upper lip, chin, chest, upper back, lower back, upper abdomen, lower abdomen, arm, forearm, thigh, and lower leg); however, this has been modified to include nine body sites because arms and legs are currently not considered to be androgen-sensitive sites. Acne is a more variable marker of hyperandrogenism. The sole presence of acne was also felt to be a potential marker for hyperandrogenism, although studies are somewhat conflicting regarding the exact prevalence of androgen excess in these patients. The sole presence of androgenic alopecia as an indicator of hyperandrogenism has been less well studied. However, it appears to be a relatively poor marker of androgen excess, unless present in the oligo-ovulatory patient. [14, 15]

Biochemical hyperandrogenism:-

Excessive androgen production with or without clinical signs is a consistent, but not universal,feature of PCOS. To satisfy current PCOS diagnostic criteria the patient should either have clinical and/or biochemical signs of hyperandrogenism. If hirsutism is unequivocally present then thecriteria are matched but other signs of hyperandrogenism such as acne, hair loss, or mild hirsutism may be more difficult to determine. The major circulating androgens are testosterone,androstenedione, dehydroepiandrosterone (DHEA), and DHEA sulfate (DHEAS).

In adult females, testosterone is the most clinically relevant circulating androgen, and has both an adrenal (25%) and ovarian (25%) contribution, but is mainly produced by peripheral conversion from circulating androstenedione. There is currently no consensus on what is the best androgen to measure or the upper cut-off consistent with PCOS but it is generally accepted that testosterone is the measurement of choice for the investigation of female hyperandrogenism. Total testosterone is not, however, invariably elevated in PCOS. The measurement of free testosterone or non-specifically bound testosterone may. Represent, more closely, the biologically active component and therefore may be of more value than totaltestosterone in the assessment of hyperandrogenism. The apparent free testosterone concentration obtained by equilibrium dialysis as well as the fraction of serum testosterone not precipitate by 50% ammonium sulfate concentration (non-sex hormone binding globulin [SHBG]-testosterone), often referred to as bioavailable testosterone, appear to represent reliable indexes of biologically readily available testosterone. Other parameters have been used without. Complete validation is the free androgen index (FAI=the ratio 100* testosterone/SHBG). [15]
The calculated FAI is by far the most widely used parameter for the assessment of female hyperandrogenism. In a recent study, Kumar et al. suggested that the prevalence of supranormal DHEAS, an androgen produced exclusively in the adrenal is approximately 20% among white and 30% among black PCOS patients. Given that an isolated increase in DHEAS is a marker of other adrenal disorders and that this weak androgen is usually associated with increased testosterone, FAI, and androstenedione in PCOS, we do not recommend that it is routinely measured in PCOS patients. [11]

Polycystic ovaries (PCO):-

Define PCO are the following: “presence of 12 or more follicles in each ovary measuring 2±9 mm in diameter, and/or increased ovarian volume (>10 mL)”. The follicle distribution should be omitted as well as the increase in stromal echogenicity and volume. Although increased stromal volume is a feature of PCO, it has been shown that the measurement of the ovarian volume is a good surrogate for the quantification of stromal volume in clinical practice. Only one ovary fitting this definition is sufficient to define PCO. If there is evidence of a dominant follicle (>10 mm) or a corpus luteum, the scan should be repeated the next cycle. A woman having PCO in the absence of an ovulatory disorder or hyperandrogeni (“asymptomatic” PCO) should not be considered as having PCOS, until more is known regarding the clinical presentation. The following technical recommendations should be highlighted:

1. Whenever possible, the transvaginal approach should be utilized, particularly in obese patients.
2. Regularly menstruating women should be scanned in the early follicular phase (cycle days 3±5).
3. Oligo-/amenorrhoeic women should be scanned either at random or between days 3 and 5 after progestin-induced withdrawal bleeding.
4. Calculation of ovarian volume is performed using the simplified formula for a prolate ellipsoid(0 .53 length 3 width 3 thickness). [15]

Insulin resistance:-

Defined as decreased insulin-mediated glucose utilization, is commonly found in the larger population (10±25%) when sophisticated dynamic studies of insulin action are performed. Hyperinsulinism and insulin resistance are also typically present in women with polycystic ovary syndrome (PCOS).

Hyperinsulinism may alter the FSH-to-LH shift, preventing the selection of a dominant follicle. Moreover, insulin seems to increase granulosa cells' sensitivity to LH and increases the production of androgens from the ovary by stimulating cytochrome P450c17α. Some studies suggest that ovarian theca cells in PCOS-affected women are more capable of converting androgenic precursors to testosterone than in normal women. Finally, and most importantly, hyperinsulinemia impedes ovulation. Such insulinresistance is worsened by physical inactivity and upper abdominal obesity.

As a consequence of the elevated LH levels, in PCOS ovaries, there is an increase in granulosa cell VEGF production.VEGF is known to be the main mediator of ovarian hyperstimulation syndrome, a grave complication of ovulation induction seen most often in women with PCOS.

Since women with PCOS are insulin resistant, they have approximately a 2-4 fold higher risk of developing diabetes compared to body mass index (BMI) similar to women without PCOS. Rather, screening for diabetes should be restricted to those at significantly elevated risk, namely those who are obese (BMI>30kg/m2) or have a family history of type 2 diabetes. Furthermore, the exact mode of screening for diabetes is not clear or universally accepted. Many groups have recommended adding an oral glucose tolerance test (OGTT) to these fasting blood tests and to evaluate the 2 h glucose level after a 75 g oral glucose challenge for glucose intolerance (WHO criteria, impaired glucose tolerance [IGT]>140±199 mg/dL).IGT has long been recognized as a major risk factor for diabetes.

There is no clear evidence that women with PCOS are at increased (3±7 times) risk of developing type2 diabetes. There are several lines of evidence suggesting that women with PCOS are also at increased risk of cardiovascular disease. Insulin-resistant states are associated with greater than normal susceptibility to coronary heart disease and women with PCOS have evidence of dyslipidemia and markers of abnormal vascular function.

Gonadotrophins:-

Abnormal gonadotrophin secretion, especially increased LH concentrations, is one of the most common findings in PCOS. Both the absolute level of circulating LH and its relationship to FSH levels are significantly elevated in PCOS women as compared with controls. This is due to an increased amplitudeand frequency of LH pulses. Elevated LH concentrations (above the 95th percentile of normal) can be observed in ~60% of PCOS women whereas the LH/FSH ratio may be elevated in up to 95% of subjectsif women who have ovulated recently are excluded.37BMI should, therefore, also be considered when Assessing whether the LH or the LH/FSH ratio is abnormally high. Based on the aforementioned data, the panel felt that the measurement of serum LH levels should not be considered necessary for the clinical diagnosis of PCOS. LH level could be useful as a secondary parameter. [22]

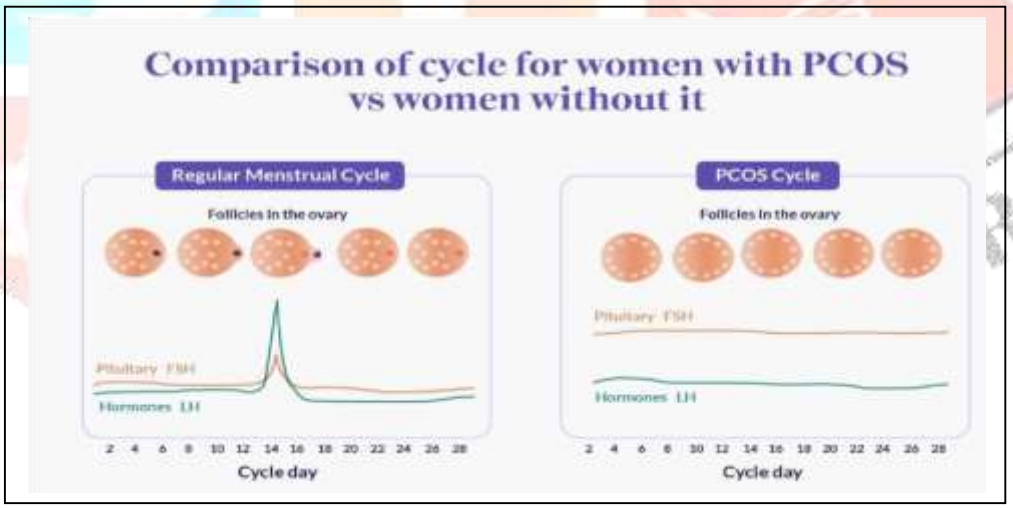


Figure No:- 4 (Comparison of cycle for women with PCOS vs women without it)

TREATMENT FOR PCOS:- Drugs used in PCOS

Clomiphene

Clomiphene Citrate:- Clomiphene is a medication used to induce ovulation.

Brand Names:- Clomid

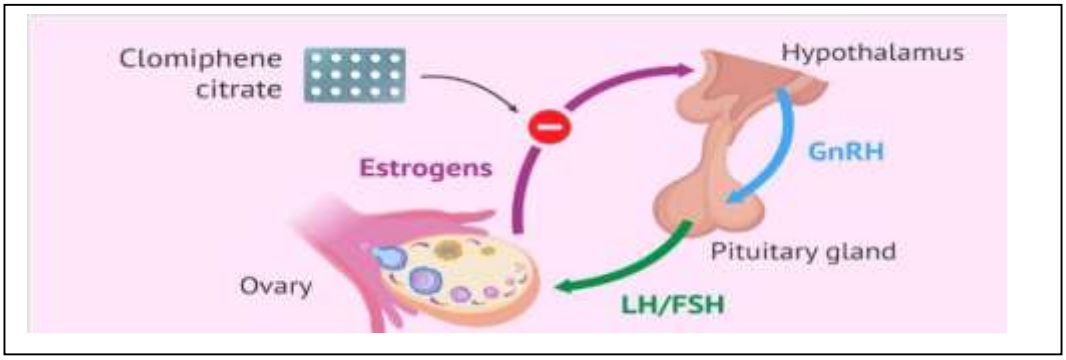
Generic Name: - Clomifene

Background:- A triphenyl ethylene stilbene derivative which is an estrogen agonist or antagonistdepending on the target tissue.

Indication:- Used mainly in female infertility due to anovulation (e.g. due to polycystic ovarysyndrome) to induce ovulation

Mechanism of action:-

Clomifene has both estrogenic and anti-estrogenic properties, but its precise mechanism of action has not been determined. Clomifene appears to stimulate the release of gonadotropins, follicle-stimulating hormone (FSH), and luteinizing hormone (LH), which leads to the development and maturation of the ovarian follicle, ovulation, and subsequent development and function of the corpus luteum, thus resulting in pregnancy. Gonadotropin release may result from direct stimulation of the hypothalamic-pituitary axis or from a decreased inhibitory influence of estrogens on the hypothalamic-pituitary axis by competing with the endogenous estrogens of the uterus,



pituitary, or hypothalamus. Clomifene has no apparent Progestational, androgenic, or antiandrogenic effects and does not appear to interfere with pituitary-Adrenal or pituitary–thyroid function. [26]

Figure No:- 5 (Mechanism of clomiphene)

***Metformin*:-**

Metformin is a biguanide antihyperglycemic used in conjunction with diet and exercise for glycemic control in type 2 diabetes mellitus. It is also used off-label for insulin resistance in polycystic ovary syndrome (PCOS).
Brand Names:- Actoplus Met, Avandamet, Fortamet, Glucophage, Glucovance, Glumetza, Glycon, Invokamet, Janumet, Jentadueto, Kazano, Kombiglyze, Komboglyze, Qternmet, Riomet, Segluromet, Synjardy, Trijardy, Velmetia, Xigduo.

Background:-

Metformin is a biguanide antihyperglycemic agent and first-line pharmacotherapy used in the management of type II diabetes. Metformin is considered an antihyperglycemic drug because it lowers blood glucose concentrations in type II diabetes without causing hypoglycemia. It iscommonly described as an "insulin sensitizer", leading to a decrease in insulin resistance and a clinically significant reduction of plasma fasting insulin levels. Another well-known benefit of this drug is modest weight loss, making it an effective choice for obese patients with type II diabetes.
Metformin was first approved in Canada in 1972 and received subsequent FDA approval in the US in 1995.
Generic Name: - Metformin.
Indication:- Metformin immediate-release formulations. Metformin is indicated as an adjunct to diet and exercise to improve glycemic control in adults and pediatric patients ≥10 years old with type 2 diabetes mellitus.

Metformin extended-release tablet (XR):-

The extended-release formulation of metformin is indicated as an adjunct to diet and exercise to improve glycemic control in adults with type 2 diabetes mellitus. Safety in children has not been determined to this date.

Metformin combination products:-

Metformin is a component of a variety of combination products with other anti-diabetic agents. It is indicated, along with diet and exercise, to improve glycemic control in adult patients with type 2 diabetes mellitus in combination with DPP-4 inhibitors (sitagliptin, linagliptin, alogliptin, or saxagliptin) in combination with SGLT2 inhibitors (canagliflozin, empagliflozin, ertugliflozin, or dapagliflozin)or combination with pioglitazone.
Mechanism of action
Metformin's mechanisms of action are unique from other classes of oral antihyperglycemic drugs. Metformin decreases blood glucose levels by decreasing hepatic glucose production (also called gluconeogenesis), decreasing the intestinal absorption of glucose, and increasing insulin sensitivity by increasing peripheral glucose uptake and utilization. It is well established that metformin inhibits mitochondrial complex I activity,

and it has since been generally postulated that its potent antidiabetic effects occur through this mechanism. The above processes lead to a decrease in blood glucose, managing type II diabetes and exerting positive effects on glycemic control. [26]

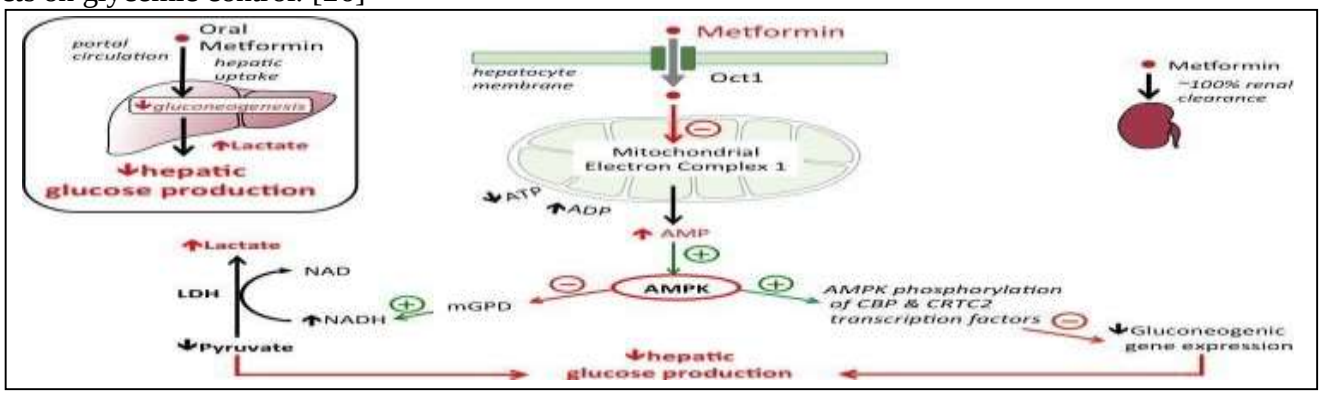


Figure No:- 6 (Mechanism of metformin)

Statins:-

Statins, also known as HMG-CoA reductase inhibitors, are a class of lipid-lowering medications that reduce illness and mortality, in those who are at high risk of cardiovascular disease. This systematicreview and meta-analysis of 9 RCTs indicated that statins could reduce the levels of androgens andimprove the cutaneous manifestations of hyperandrogenism in women with PCOS.

Antiandrogens

Antiandrogens such as spironolactone, CPA, or flutamide act by competitive inhibition of androgen-binding receptors or by decreasing androgen production. Spironolactone, which is an aldosterone antagonist, is a dose-dependent competitive inhibitor of the androgen receptor and can also inhibit 5- α-reductase activity Their mechanism of action addresses many PCOS symptoms such as hirsutism, oily skin and acne, and scalp hair loss. These drugs can cause problems during pregnancy, so do not take them if you want to get pregnant or are pregnant. [26]

Oral Contraceptive:-

Oral contraceptives (OCs; with combined estrogen and progestin) are the most commonly used treatment for regulating menstrual periods in women with PCOS. OCs protect the woman from endometrial (uterine) hyperplasia or cancer by inducing a monthly menstrual period. Some pills, such as Loestrin, have lower estrogen levels. These low estrogen levels can reduce the severity of some side effects but may also be less effective against symptoms of PCOS.

Best birth control pills for PCOS:-

Mircette, Natazia ,Nordette ,Lo/Ovral ,Ortho-Novum Ortho Tri-Cyclen ,Yasmin.

Inositol:-

Inositols act as second messengers for insulin, and their deficiency contributes to the various features ofPCOS. Correction of MI and DCI deficiency simultaneously, by using MDI therapy, may help in alleviating metabolic, menstrual/ovulatory, and cutaneous hyperandrogenic features of PCOS. Various types of inositol have been shown as an effective primary treatment for women with polycystic ovarian syndrome (PCOS). With emerging research on its efficacy, this supplement can be very beneficial in treating symptoms of PCOS. For the treatment of polycystic ovarian syndrome (PCOS), myo-inositol is taken in the range of 200- 4,000mg once daily before breakfast, the higher dose seems to be used more often and seems more effective. [24]

Aromatase inhibitors:-

PCOS is the most common cause of infrequent or absent menstrual periods and affects about 5% to 20% of women worldwide. It often causes anovulatory infertility (infertility related to the inability to ovulate). Aromatase inhibitors (AIs) are used to make ovulation happen. By lowering estradiol levels; aromatase inhibitors reduce the negative feedback and thus increase pituitary gonadotropin output. As a result, ovarian function increases.

Research has shown that Letrozole can help to significantly increase pregnancy and live birth rates for individuals with PCOS. Those with PCOS commonly use Letrozole to induce ovulation in combination with different fertility treatments (like IUI) to increase their chances of getting pregnant. [28]

Medroxyprogesterone acetate:-

Oral contraceptive pills or medroxyprogesterone acetate (MPA) are commonly used drugs for the management of conditions associated with PCOS, such as hirsutism and oligomenorrhea. The androgenic properties of MPA and other progestins in the oral contraceptive cells may decrease insulin sensitivity. Progesterone slows the midcycle pulses of LH (and GnRH), which are too rapid in people with PCOS. Taking cyclic progesterone when the body isn't producing its own (due to lack of ovulation) makes the LHpulses slow down. [28]

Glucagon-like peptide -1 receptor analog:-

Visceral fat dysfunction is an important factor in the onset of PCOS. GLP-1 receptor agonist is a glucagon-like peptide 1 analog, which is related to improving blood sugar control, weight loss and appetite suppression, and reducing cardiovascular risk.The GLP-1RAs have been shown to significantly improve glycemic parameters and reduce bodyweight. These agents work by activating GLP1 receptors in the pancreas, which leads to enhanced insulin release and reduced glucagon release responses that are both glucose-dependent-with a consequent low risk for hypoglycemia. [28]

MANAGEMENT OF PCOS:-

Modification in lifestyle:-

Over half of all PCOS sufferers are overweight or obese, so PCOS patients are primarily recommended to reduce weight since a good, balanced diet combined with regular exercise can raise their metabolism, improve insulin sensitivity, and help them lose weight safely. Patients of PCOS have hormonal imbalances, high blood cholesterol levels, and are obese. It is critical to understand that working out alone will never be enough to help them lose weight. It is more important to have a healthy diet. For Indian women, diet is seldom a priority. A healthy diet should be high in fiber and protein (1 g/kg body weight). A 30% calorie deficit, or 500 to 750 kcal per day (1200 to 1500 kcal per day), should be stated. According to various studies, overweight people can lose weight, and PCOS females with infertility hadirregular ovulation and more excellent responsiveness to ovulation induction drugs, resulting in higher pregnancy and live birth rates. According to research, reducing up to 5% of one's initial weight can help restore regular menstruation and boost the reaction to ovulation and reproductive medications. [21]

Medication to lose weight:-

1.Orlistat
2.Sibutramie
3.Rimonabat
4.Naltrexone/bupropion

FOOD INTAKE THAT SHOULD BE AVOIDED IN PCOS:-

SUGARY FOODS: Many PCOS sufferers have higher insulin levels than normal. This can cause difficulty in losing weight. Eating fewer sugars and simple carbohydrates can help them to lose weight and lower the risk of diabetes.

FOODS MADE WITH WHITE FLOUR: White flour is a simple carbohydrate that most PCOS sufferers should avoid. Breads, bagels, cereals, muffins, cupcakes, and other baked goods are common sources of white flour

SODIUM-RICH FOODS: PCOS sufferers should limit their sodium intake to a maximum of 2,300mg/day or be cautious of 1,500mg daily. Skip high-sodium foods like smoked meats, canned vegetables, chips, sauces, salted nuts, and canned soups.

FATTY FOODS: PCOS sufferers should avoid saturated fats, meats, fat-free dairy, fat-free dressing, white meat, and skinless poultry. Instead of frying food, steam, broil, bake, grill or microwave them to avoid excess oil. [19]

PCOS DIET PLAN: - One of the most common side effects of this health condition is uncontrolled weight gain. However, a typical "Low-Fat" weight loss diet like the simple 1200-calorie diet plan is not effective enough to promote weight loss in women suffering from PCOS because in this case, weightgain is a result of high insulin levels that promote fat storage in the body.

Table No:- 3 (Diet plan of PCOS)

Breakfast	Avoid carbs and sugary cereals.Pair proteins with healthy fats. Make breakfast your heaviest meal	Example: - Scrambledeggs, Sauteed veggies, Whole wheattoast, and avocado.
Lunch	Pack lunch to avoid bad choices. Eat complex carbs.	Example: -Brown rice,Roasted chicken, Broccoli Spinach.
Snacks	Satisfy cravings withcrunchy foods. Choose high-fiber snacks Drink half your body weight inounces of water daily.	Example: -Apple slices, Almondbutter, Fresh strawberries.
Dinner	Add a variety of colorful vegetables. Choose lean meats. Eat before 7pm.Make dinner the lightest meal.	Example: -Salmon with stir-fried greenbeans, Broccoli, Carrots, Mushrooms .

VITAMIN D:-

There is increasing evidence suggesting that PCOS affects the whole life of a woman, can begin in utero in genetically predisposed subjects, manifests clinically at puberty, and continues during the reproductive years [16]. Vitamin D insufficiency or inadequacy affects 45-90% of reproductive-age women. According to research, vitamin D insufficiency was associated with a substantial reduction in ovulation rate, pregnancy rate, and the chance of a live delivery in PCOS women receiving ovarian stimulation for infertility. Patients with polycystic ovarian syndrome, ovulation dysfunction, and metabolic disorders maybenefit from vitamin D medication. To make firm conclusions on the effect of vitamin D supplementationon female reproductive health, randomized, prospective, and controlled studies are required. [18]

HOME REMEDIES TO CURE PCOS:-

Individuals can add the below-mentioned foods to their diet. Methi, Flaxseeds, Cinnamon (Dalchini), Holy basil (Tulsi), Honey, Bitter gourd (Karela), Indian Gooseberry (Amla). PCOS Prevention In Young FemalesRegular exercise, healthy food, and weight control are the key treatments for PCOD.

- Try to fit in moderate activity and/or vigorous activity often. Walking is a great exercise thatmost people can do.
- Eat heart-healthy foods. This includes lots of vegetables, fruits, nuts, beans, and whole grains. Itlimits foods that are high in saturated fat, such as meats, cheeses, and fried foods.
- Most women who have PCOD can benefit from losing weight. Even losing 10 lb. (4.5 kg) mayhelp get your hormones in balance and regulate the menstrual cycle.
- Quit smoking.
- Physicians also may prescribe birth control pills to reduce symptoms, Metformin helps to regulate
- Menstrual cycles, or fertility medicines if a woman having trouble getting pregnant.
- It may take a while for treatments to help with symptoms such as facial hair or acne. Patients canuse over-the-counter or prescription medicines for acne.
- It can be hard to, deal with having PCOD. If the patients feel sad or depressed, it may help to talk to a counselor or to other women who have PCOD or with a similar clinical condition. [19]

YOGA

Several studies have been undertaken to assess the impact of yoga, the ancient art of yoga that builds harmony between our mind and body, on PCOS. These studies along with first-hand experiences of women who have been practicing yoga for PCOS have shown a positive correlation between yoga and Their alleviated symptoms. Yoga poses for PCOS have been shown to have physical and mental benefits inbolstering your health & wellness. [25]

Asanas which are good for PCOS patients:-

1.Bhadrasana (Butterfly pose)
2.Malasana (Garland pose)
3.Sarvangasana (Reverse body)

Here is how yoga for PCOS helps you.

- Yoga asanas for PCOS help in abdominal compression, improving blood circulation and metabolism.
- Yoga asanas for PCOS help in decreasing the levels of cortisol.
- Yoga for PCOS helps in decreasing blood sugar and insulin resistance.
- Yoga for PCOS helps in regulating menstrual cycles.
- Yoga for PCOS works wonders in reducing anxiety and stress.
- Yoga poses for PCOS helps in decreasing painful inflammation.
- Yoga for irregular periods and PCOS may help in reducing androgens.
- Yoga asanas for PCOS help in reducing weight. This helps in dealing with feelings of body shame and disappointment and inducing self-acceptance.

Yoga asanas for PCOS help in reducing weight. This helps in dealing with feelings ofbody shame and disappointment and inducing self-acceptance

COMPLICATIONS IN PCOS:-

Many studies have been performed comparing pregnancy outcomes in women with PCOS vs controls.

MULTIPLE PREGNANCIES: - Multiple pregnancies are the most important cause of the increased perinatal morbidity observed following fertility treatments, with special regard to women with PCOS affected by ovulatory infertility. Most of the risk of pregnancy complications is due to preterm delivery rates of multiple births.

MISCARRIAGE: - It is still debated whether women with PCOS have an increased risk of miscarriage compared to women without fertility disorder.

PREGNANCY-INDUCED HYPERTENSION AND PRE-ECLAMPSIA;- Pregnancy-induced hypertension in women with PCOS. Women with PCOS also represent a 3-4-fold increased risk of developing pre-eclampsia during pregnancy.

GESTATIONAL DIABETES MELLITUS: - It is the most commonly described pregnancy complication in women with PCOS. Its early diagnosis is crucial and its careful treatment significantly reduces the incidence of related maternal and neonatal complications. The risk of GDM is about 3 times higher in women with PCOS. [4,20]

RISK FACTOR IN PREGNANCY

This is perhaps one of the most discussed topics among women who are trying to get pregnant. While there is an increased awareness about the same, many women may not notice that they have it till they try to conceive. While PCOS can cause complications in conception, pregnancy, and childbirth, it is now possible to treat the symptoms and conceive and deliver babies safely with the right steps and doctor's suggestions. [30]

1. What happens to PCOS during pregnancy?

PCOS is not a fully treatable disease and thus doesn't fully go away. However, with some lifestyle changes and medications, one can manage the symptoms well. During pregnancy, it's best to keep a close eye on your health due to the health risks of PCOS and pregnancy. Follow the doctor's advice and stay healthy to minimize the effects of PCOS on pregnancy.

2. How does PCOS affect early pregnancy?

PCOS causes many diseases in women like diabetes, high blood pressure and cholesterol, etc. which in turn can affect the unborn child. Women with PCOS are also at a higher risk of miscarriage but with the proper knowledge, precautions, and doctor's guidance it is possible to have safe pregnancy and delivery even with PCOS.

3. Can PCOS cause birth defects?

While studies do not offer a conclusive result on whether PCOS causes birth defects, the other symptoms and side effects of PCOS can negatively impact your or your child's health during childbirth. Thus it's best to know more about PCOS and follow all the doctor's suggestions on the same.

4. What is the success rate of pregnancy with PCOS?

While many women struggle to get pregnant with PCOS, most women can do so with some lifestyle changes like losing weight, avoiding smoking and alcohol, and consulting doctors for other changes and medication.

5. What happens if PCOS is left untreated?

If left untreated, PCOS can worsen the other symptoms thus leading to poor health and increased healthrisks caused by high blood pressure and cholesterol (which in turn affect cardiovascular health), diabetes, sleep apnea, etc.

2. CONCLUSION:-

The reviewed article is based on, PCOS which commonly affects women of reproductive age so appropriate advice regarding the impact of lifestyle, obesity, and fertility should be offered. Women withPCOS are at increased risk of adverse pregnancy and birth outcomes and increased surveillance during pregnancy. Adequate support should be offered life lifelong lifestyle modification aiming for a target of healthy weight. Women should be informed of the increased risks of pregnancy complications and the potential for adverse outcomes for their offspring. The cause of PCOS is unclear, but early diagnosis can help relieve systems and reduce the risk of complications.

3.REFERENCE:-

1. Armanini, D., Boscaro, M., Bordin, L. and Sabbadin, C., 2022. Controversies in the Pathogenesis, Diagnosis, and Treatment of PCOS: Focus on Insulin Resistance, Inflammation, and Hyperandrogenism. *International Journal of Molecular Sciences, 23*(8), p.4110
2. Artini, P.G., Di Berardino Artini, P.G., Di Berardino, O.M., Simi, G., Papini, F., Ruggiero, M., Monteleone, P. and Cela, V., 2010. Best methods for identification and treatment of PCOS. *Minerva ginecologica, 62*(1), p.33.
3. Shrivastava, V., Batham, L., Mishra, S. and Mishra, A., 2019. An Integrated Traditional Therapy Approach Involving Yagya Therapy In Patient WithSymptoms Similar to Crohn's Disease. *Interdisciplinary Journal of Yagya Research, 2*(2), pp.11-19.
4. Satyanarayana, V., Reddy, D.B., Prathima, K., Prasanthi, G. and Sowmya, L., 2019. A review on PCOD in pregnant women. *World Journal of Current Medical and Pharmaceutical Research*, pp.113-115.
5. Mancini, V. and Pensabene, V., 2019. Organs-on-chip models of the female reproductive system. *Bioengineering, 6*(4), p.103.
6. De Ziegler, D., Borghese, B. and Chapron, C., 2010. Endometriosis and infertility: pathophysiology and management. *The Lancet, 376*(9742), pp.730738.
7. Begum, M., Das, S. and Sharma, H.K., 2016. Menstrual disorders: causes and natural remedies. J Pharm Chem Biol Sci, 4(2), pp.307-20.
8. Freeman, E.W., Sammel, M.D., Lin, H., Gracia, C.R., Pien, G.W., Nelson, D.B. and Sheng, L., 2007.

Symptoms associated with menopausal transition and reproductive hormones in midlife women. *Obstetrics & Gynecology*, *110*(2 Part 1), pp.230
9. https://www.aafp.org/pubs/afp/issues/2016/0715/p106/jcr:content/root/aafp-article- primary-content. container/aafp_article_main_par/aafp_tables_content1.enlarge.html
10. Diamanti-Kandarakis, E. and Christakou, C.D., 2009. Insulin resistance in PCOS *Diagnosis and management of polycystic ovary syndrome*, pp.35-61.
11. Marshall, J.C. and Dunaif, A., 2012. Should all women with PCOS be treated for insulin resistance? *Fertility and sterility*, 97(1), pp.18-22.
12. Venkatesan, A.M., Dunaif, A. and Corbould, A., 2001. Insulin resistance in polycystic ovary syndrome: progress and paradoxes. *Recent progress in hormone research*, *56*, pp.295-308.
13. Firdose, K.F. and Shameem, I., 2016. An approach to the management of polycystic ovarian disease in Unani system of medicine: A review. *Int j appl res*, *2*(6), pp.585-90.
14. Anjali, C.S., George, M., Das, P. and Soji, S., 2019. PCOD IN FEMALE REPRODUCTIVE
15. Williams, T., Mortada, R. and Porter, S., 2016. Diagnosis and treatment ofpolycystic ovary syndrome.*American family physician*,*94*(2),pp.106-113.
16. Sirmans, S.M. and Pate, K.A., 2014. Epidemiology, diagnosis, and management ofpolycystic ovary syndrome. *Clinical epidemiology*, *6*, p.1.
17. Karakas, S.E., 2017. New biomarkers for diagnosis and management of polycysticovary syndrome. *Clinica Chimica Acta*, *471*, pp.248-253.
18. Sheehan, M.T., 2004. Polycystic ovarian syndrome: diagnosis and management.*Clinical Medicine & Research*, *2*(1), pp.13-27.
19. Al Wattar, B.H., Fisher, M., Bevington, L., Talaulikar, V., Davies, M., Conway,G. and Yasmin, E., 2021. Clinical practice guidelines on the diagnosis and management of polycystic ovary syndrome: a systematic review and quality assessment study. *The Journal of Clinical Endocrinology & Metabolism*, *106*(8), pp.2436-2446.
20. Spritzer, P.M., 2014. Polycystic ovary syndrome: reviewing diagnosis and management of metabolic disturbances. *Arquivos Brasileiros de Endocrinologia &Metabologia*, *58*, pp.182187
21. Roya, R., Mohammad Akbar, A., Wajeeda, T., Avinash, B., Humaira, M., Avvari, B.B., Ayapati, M.G., Vikram, A.A., Taalia, N.A. and Mohammed, S.A., 2021. An Indian EvidenceBased Study of Prevalence, Phenotypic Features, LifestyleModifications of Polycystic Ovarian Syndrome Patients. J. Gynecol. Women's Health, 21, p.556069.
22. Setji, T.L. and Brown, A.J., 2014. Polycystic ovary syndrome: update on diagnosis and treatment. The American journal of medicine, 127(10), pp.912-919.
23. https://www.news-medical.net/health/Recent-Research-into-Polycystic-Ovary- Syndrome.aspx
24. Carmina, E., Oberfield, S.E. and Lobo, R.A., 2010. The diagnosis of polycystic ovary syndrome in adolescents. *American journal of obstetrics and gynecology*, *203*(3), pp.201-e1.
25. Azziz, R., Carmina, E., Dewailly, D., Diamanti-Kandarakis, E., Escobar-Morreale,H.F., Futterweit, W., Janssen, O.E., Legro, R.S., Norman, R.J., Taylor, A.E. and Witchel, S.F., 2009. The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: the complete task force report. *Fertility and sterility*, *91*(2), pp.456-488.
26. Eshre, R. and ASRM-Sponsored PCOS Consensus Workshop Group, 2004. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). *Human Reproduction (Oxford, England)*, *19*(1), pp.41-47.
27. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group, 2004. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). *Human reproduction*, *19(1)*, pp.41-47.
28. Kahsar-Miller, M.D., Nixon, C., Boots, L.R., Go, R.C. and Azziz, R., 2001. Prevalence of polycystic ovary syndrome (PCOS) in first-degree relatives of patients with PCOS. *Fertility and sterility*, *75(1)*, pp.53-58
29. Rosenfield, R.L. and Ehrmann, D.A., 2016. The pathogenesis of polycystic ovary syndrome (PCOS): the hypothesis of PCOS as functional ovarian hyperandrogenism revisited. *Endocrine reviews*, *37(5)*, pp.467-520.
30. Fauser, B.C., Tarlatzis, B.C., Rebar, R.W., Legro, R.S., Balen, A.H., Lobo, R., Carmina, E.,Chang, J., Yildiz, B.O., Laven, J.S. and Boivin, J., 2012. Consensus on women's health aspects of polycystic ovary syndrome (PCOS): the Amsterdam ESHRE/ASRM-Sponsored 3rd PCOS Consensus Workshop Group. *Fertility and sterility*, *97(1)*, pp.28-38.
31. Kandaraki, E., Chatzigeorgiou, A., Livadas, S., Palioura, E., Economou, F., Koutsilieris, M., Palimeri, S.,

Panidis, D. and Diamanti-Kandarakis, E., 2011. Endocrine disruptors and polycystic ovary syndrome (PCOS): elevated serum levels of bisphenol A in women with PCOS. *The Journal of Clinical Endocrinology & Metabolism, 96(3)*, pp.E480-E484.

32. De Groot, P.C., Dekkers, O.M., Romijn, J.A., Dieben, S.W. and Helmerhorst, F.M., 2011. PCOS, coronary heart disease, stroke and the influence of obesity: a systematic review and meta- analysis. *Human reproduction update, 17(4*), pp.495- 500.
33. De Leo, V., Musacchio, M.C., Cappelli, V., Massaro, M.G., Morgante, G. and Petraglia, F.J.R.B., 2016. Genetic, hormonal and metabolic aspects of PCOS: an update. *Reproductive Biology and Endocrinology, 14(1)*, pp.1-17.
34. Cronin, L., Guyatt, G., Griffith, L., Wong, E., Azziz, R., Futterweit, W., Cook, D. and Dunaif, A., 1998. Development of a health-related quality-of-life questionnaire (PCOSQ) for women with polycystic ovary syndrome (PCOS). *The Journal of Clinical Endocrinology & Metabolism, 83(6)*, pp.1976-1987.
35. Diamanti-Kandarakis, E., Kandarakis, H. and Legro, R.S., 2006. The role of genes and environment in the etiology of PCOS. *Endocrine, 30(1)*, pp.19-26.
36. Ning, N., Balen, A., Brezina, P.R., Leong, M., Shoham, Z., Wallach, E.E. and Zhao, Y., 2013. How to recognize PCOS: results of a web-based survey at IVF- worldwide. com. *Reproductive biomedicine online, 26(5)*, pp.500-505.