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RESEARCH ARTICLE

ANTHROPOMETRIC MEASUREMENTS IN WOMEN WITH POLYCYSTIC OVARY SYNDROME IN SAUDI ARABIA.

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Abstract

Background: Polycystic Ovary Syndrome is a common endocrine disorder that is characterized by the presence of chronic anovulation, excess levels of androgen as well as polycystic ovary on ultrasonographic imaging.

Objectives: To determine the causes, complications and clinical features of polycystic ovary syndrome. To assess the anthropometric measurements of obesity in polycystic ovary syndrome Saudi women and to provide treatment for the associated obesity.

Review of Literature: Polycystic Ovary Syndrome involves interactions between different components such as the anterior pituitary gland, hypothalamus, ovaries, suprarenal glands and peripheral fatty tissues. It seems to be a multifactorial disease. It presents with different manifestations and it can affect the metabolic and reproductive functions as well as the cardiovascular system. Abdominal obesity can initiate the pathogenesis of Polycystic Ovary Syndrome and the opposite is true. Clinical features, laboratory tests and ultrasound imaging are the clinical diagnosis. There is a high incidence rate within Saudi population due to increasing obesity. It has a significant effect on anthropometric body profiles of Saudi women. However, lifestyle modifications and pharmacological therapies have an essential role in management of obesity related to this syndrome.

Conclusion: The etiology of Polycystic Ovary Syndrome is multifactorial and is linked to long term complications that present with cardinal features. It is significantly associated with high anthropometric findings in Saudi Arabian women, but lifestyle interventions and pharmacotherapy are effective in losing weight.

Introduction:-

Different studies have been proposed that Polycystic Ovary Syndrome (PCOS) is a clinical condition that is characterized by the presence of chronic anovulation, excess levels of androgen as well as polycystic ovary on ultrasonographic imaging(1). It is the commonest endocrine disorder that affect 5-7 % of women at the reproductive age also, it impacts the quality of life as a consequences it is associated with significant morbidity and mortality and its prevalence in Saudi Arabia is 53.7%(2,3).

Since the first description of PCOS by Stein and Leventhal in 1935(1) the etiology of this Syndrome remains unclear , but it seems to be a multifactorial disease and among the common causes that play a significant role are increasing

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in insulin resistance which presents in 85 % of affected women , high androgen levels in 60-80 % ,genetic factors and family history and vitamin D deficiency(4).

Previous studies suggested that obesity and overweight are frequently seen in PCOS patients(3). It is defined as increasing of the fat accumulation in adipose tissues within the body and the body mass index(BMI) more than or equal to 30 Kg / m^2 (5). Body mass index and waist circumference are the critical anthropometric measurements for obesity(6). In addition, Saudi Arabia is one of the most countries of high prevalence of obesity that found in 7 persons out of 10 and it is significantly affect the females more with a prevalence of 58.7 % compared to the males(7). Many studies reported that, losing weight thought out life style modifications and pharmacological therapies can play an essential role in management and treatment of obesity that associated with polycystic ovary syndrome(8).

The aims of the current study are to determine the causes, complications and clinical manifestations of polycystic ovary syndrome as well as to assess the anthropometric parameters of obesity among Saudi women who have been diagnosed with PCOS and finally to provide proper treatment for the associated obesity.

THE OBJECTIVES OF THIS STUDY ARE

1. To determine the causes, complications and clinical features of polycystic ovary syndrome.
2. To evaluate the anthropometric measurements of obesity in Saudi women with polycystic ovary syndrome.
3. To provide treatment for obesity that associated with polycystic ovary syndrome.

Review of literature

1. Definition of PCOS

It is believed that Polycystic Ovary Syndrome is a complex endocrinal disease that involves interactions between different body components such as the anterior pituitary gland, hypothalamus, ovaries, suprarenal glands and peripheral fatty tissues(9). It was initially defined by Stein and Leventhal in 1935 as increasing in ovarian diameter, presence of numerous follicular cysts and increasing in the thickness of the capsule(9).

In 1990, the National Institute of Health (NIH) defined the PCOS as the only presence of excess androgen with anovulation, but in 2003, the European Society of Human Reproduction and Embryology/American Society for Reproductive Medicine (ESHRE/ASRM) in Rotterdam established a standard international criteria for diagnosis of PCOS(9). This criteria states that at least two of the following three should be present in those patients: chronic anovulation, hyperandrogenism and existence of 12 or more follicles with 2 to 9mm of diameter or the volume in at least one ovary is greater than 10 cm^3(9).

Later in 2004, the Androgen Excess Society (AES) considered the presence of high androgen levels as an important factor in causing Polycystic Ovary Syndrome and must be accompanied with amenorrhea or polycystic ovary on ultrasonographic imaging(9).

2. Causes of PCOS

Increase insulin resistance

Several studies found a relative connection between increasing in insulin levels which is known as hyperinsulinemia as responding to insulin resistance with development of Polycystic Ovary Syndrome by several different mechanisms(11). Elevated androgen levels can lead to changes of skeletal muscle fibers into type IIb which is more likely to be insensitive to insulin furthermore, insulin affects the process of conversion of cholesterol to the steroid hormones in adrenal glands by causing inhibition and deficiency of cortisone reductase enzyme which helps in transformation of cortisone to its active form cortisol so that gives rise to excess generation of androgen from the adrenal glands(11).

Also, it has been reported that it can result in decrease production of sex hormone binding globulin in the liver which favors facilitating the androgen action(11).When hippocampus activates are inhibited, indirectly Insulin causes the hypothalamus to secret corticotropin releasing hormone that leads to stimulation of anterior pituitary gland to produce adrenocorticotropic hormone which influences the adrenal cortex to secrets more androgen, by this way insulin enhances the action of Hypothalamus-Pituitary-Adrenal axis(HPA)(11).

In addition, insulin can be a synergism by co-acting with luteinizing hormone(LH) to rises the intracellular levels of cyclic adenosine monophosphate who stimulates the steroidogenic acute regulatory protein that converts the cholesterol to active steroids known as steroidogenesis (11).Women who have Polycystic ovary Syndrome with insulin resistance can progress to pancreatic and beta cells dysfunction and subsequent development of diabetes mellitus type 2(12). For theses reasons, authors believe that insulin resistance has an adverse effects on the metabolism of lipids, protein regulation and androgen secretions(12).

2.1 *High androgen levels*

Studies demonstrated that excess androgen in this syndrome originates from the ovaries while other studies found that it arises from the adrenal glands, but still another studies approve that hyperandrogenism developed from participation of adrenal glands along with the ovaries as both of them originate from the same embryonic tissues from the mesonephric cells(13).

After transforming of androgen into estrogen from the adrenal glands within the body organs such as the liver, it causes the pituitary gland to release luteinizing hormone which is sequentially leads to secretion of androgen from the theca cells of the ovaries and decreasing the levels of follicle stimulating hormone(FSH) which assists in failure of ovulation and development of follicles(4,13).

High dehydroepiandrosterone levels indicate that overproduction of androgen is from the adrenals source(14). The follicles that found in PCOS women presented with enlargement and hyperplasia of theca interna cells that result in increased numbers of layers consisting of differentiated steroidogenic cells than the numbers of layers observed in women without PCOS which give rise to more androgen synthesis(14). In addition, increasing the androgen levels cause negative feedback stimulation of hypothalamus and more releasing of gonadotropin thus, causing secretion of more androgen from the ovaries and the viscous cycle will be repeated (4).

2.2 *Genetic factors and Family history*

It has been demonstrated that PCOS can emerge from mutations or defects in genes that encode for proteins involved in production and modulation of androgen hormone as well as in insulin regulation such as CYP 11a, CYP 17 genes(15). The mode of transmission or inheritance is found to be an autosomal dominant associated with defects in a single gene and presents with high rates in identical twins than non-identical(16).

2.3 *Vitamin D deficiency*

Vitamin D deficiency in Saudi Arabia has been typically associated with bone and insulin-resistant diseases but narrow data are available to prove the connection(17). In this condition, the blood sugar levels may increase that leads to diabetes and excessive amount of insulin causes high androgen levels that may lead to development of PCOS in women(17).

3. Clinical diagnosis of PCOS

3.1 *clinical features*

According to the clinical features of PCOS, a cross sectional study conducted on a total of 183 Saudi participants; 93.44 % have hirsutism which is higher than the other features, 80.33% with acne, 68.31% with amenorrhea and 20.77% with infertility(18).

Hirsutism is the presence of numerous small hairs in certain body parts that are normally contain less amount of hairs such as the face or the abdomen(19). It has been found that this hairs grow because of excessive androgen that binds to androgen receptors thus, provokes changes in the hair follicles(19).

Another feature is the presence of acne which results because the androgen sends signals to the sebaceous glands particularly in the forehead that lead to the growth of sebocytes to produce sebum and therefore appearance of acne(19).

Also, alopecia which means loss of hairs because of increased levels of 5α reductase enzyme that converts the testosterone into dihydrotestosterone which acts on androgen receptors to transforms the big hair follicles into small follicles(20).

Moreover, PCOS women present with amenorrhea that refers to a condition in which the menstrual bleeding is absent for more than three months and this is usually a secondary amenorrhea and it is characterized by thick, pale and large polycystic ovaries(21). Some studies believe that elevated levels of luteinizing hormone in PCOS women stimulates the granulosa cells to secrete anti-Mullerian hormone that interferes with action of follicle stimulating hormone causing inhibition of development and growth of ovarian follicles, so it is responsible for anovulation in these women which prevents the follicles from releasing a mature egg that causing amenorrhea and infertility(22).

3.2 *Laboratory investigations*

Different studies illustrated that the laboratory investigations must look for insulin resistance and hyperandogenism for evidential supporting of Polycystic Ovary Syndrome with excluding out any other diseases that resemble some of the features of PCOS for example, thyroid dysfunction, Cushing Syndrome and tumors of adrenal glands or ovaries(23).

For evaluation of insulin resistance, they found that mentoring of fasting glucose to insulin ratio is a simple test for measurement and it should be less than 4.5 in polycystic ovarian syndrome women with obesity and normal glucose levels(23). In addition, it is enough to measure fasting glucose level which is equal or greater than 110 mg/dl plus fasting lipid profiles such as triglycerides of equal or more than 150 mg/dl and high density lipoprotein of less than 50 mg/dl. In contrast, assessment of two hour glucose tolerance test is the best marker for evaluating insulin resistance than using fasting glucose test(23).

In PCOS patients with impaired glucose tolerance, it ranges from 140 to 199 mg/dl and whom with type 2 diabetes mellitus, it equals or greater than 200 mg/dl(23). On the other hand, measuring blood free testosterone is a strong indicator for excess androgen which equals or less than 150 ng/dl in PCOS women, but higher values more than 200 ng/dl are suggestive of tumors in adrenal glands or ovaries(23).

Also, levels of dehydroepiandrosterone sulfate (DHEA-S) might be normal or little bit elevated and levels of sex hormone binding globulin(SHBG) are low in PCOS patients(23). Furthermore, measuring the concentrations of hormones other than androgen are useful in diagnosis of PCOS such as low levels of follicle stimulating hormone and high levels of luteinizing hormone or luteinizing hormone/follicle stimulating hormone ratio equals or more than 2.0(23).

3.3 *Ultrasound imaging*

Researches argued that the principal goal for using ultrasound imaging in Polycystic Ovary Syndrome is to rule out the presence of multiple ovarian cysts. According to the (ESHRE/ASRM) criteria in Rotterdam, the existence of 12 or more follicles with a diameter of 2-9 mm or the volume in at least one ovary is greater than 10 cm^3 is appropriate to confirm the presence of polycystic ovaries(24).

They also suggested that pelvic ultrasound scan which consists of two types; transabdominal and transvaginal ultrasounds is used for not just detecting the polycystic ovaries, but also for examining ovarian tumors and any sexual abnormalities(24). It is proposed that transvaginal ultrasound is more preferred technique for imaging because it is more comfortable and it provides an accurate visualization for the internal structures in particular, the obese patients while transabdominal ultrasound needs for adequately filling the bladder(24).

However, pelvic ultrasound can show a peripheral follicular distribution with stromal hyperechogenicity which are the key features on ultrasound in PCOS patients **Figure1,2**(3). In a cross sectional study on 201 Saudi females these ultrasonic features are present in 97 (89.8%) of the total cases(3).

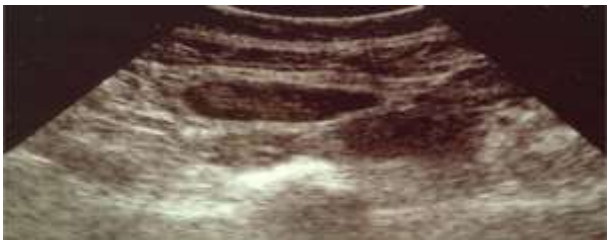

Figure 1: pelvic imaging of patient with light echogenicity of the stroma with more than 12 follicls and with peripheral distribution(3).

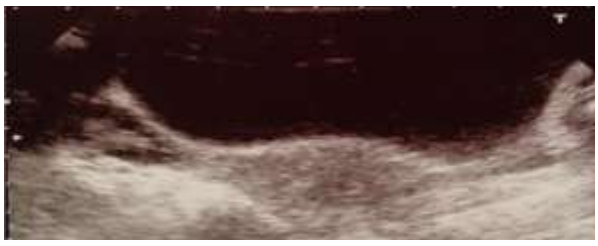

Figure 2: transabdominal imaging of the right ovary with a diameter of more than 2mm with hyperechogenic stroma(3).

4. Complications of PCOS

4.1 *Disorders in menstrual cycle*

Studies suggested that irregular menstrual cycles attributed to chronic oligoovulation or infrequent ovulation with other anomalies including endometrial persistent stimulation that increases the liability of hyperplasia and bleeding from the uterus(25).

4.2 *Type2 diabetes mellitus*

Studies showed that the incidence of type 2 diabetes mellitus over the PCOS women ranges between 7.5% and 15%(26). The risk of having Type 2 diabetes mellitus with PCOS is significantly associated with insulin resistance that causes impairment in insulin receptors, defective inhibition in the process of gluconeogenesis and abnormalities in metabolism of glucose along with deficiency in secretion of insulin secondary to dysfunction of beta cells of pancreas(27).

4.3 *Central obesity*

It is found that women with PCOS are more likely to develop central obesity compared to the healthier women(28). Increasing the levels of triglycerides(TG) up to 26mg/dl with decreasing the amounts of high density lipoproteins(HDL) into 6mg/dl are strongly correlate with insulin resistance because it can affect the lipoproteins within the body(28). Moreover, adiponectin which is a hormone with anti-inflammatory effects that play a major role in lipids, glucose metabolism and insulin regulation and it is linked to the presence of obesity, hyperglycemia and insulin resistance when it is founded in low levels(29). In addition, ApoC-1 which suppresses the uptake of triglycerides by the liver receptors which lead to atherosclerosis is higher in women with PCOS than the healthy women(30).

4.4 *Cardiovascular disease*

It is reported that hyperinsulinemia along with insulin resistance can disturb the smooth muscle cells leading to their hypertrophy and subsequent decreasing the diameter of the blood vessels also, it can activate the renin angiotensin aldosterone system that influences the levels of sodium in the blood leading to its retention and causeing hypertension(31). There is a high risk for development of ischemic heart disease in PCOS women because of increased blood pressure and serum lipids which cause more thickness in the tunica intima and tunica media layers of the coronary blood vessels(32).

4.5 *Cancer*

Studies demonstrated that estrogen stimulates the endometrial cells proliferation and mitotic divisions in contrast to the progesterone which induces the secretory phase of menstrual cycle and break down of endometrium(33). Hence, prolonged exposure to androgen which is finally converted into estrogen in the peripheral tissues and decreasing in progesterone levels allow uncontrolled endometrial cells growth and divisions and eventually cancer development(33).

5. Management of obesity in PCOS

5.1 *Life style modification*

According to lifestyle interventions, they include modification of dietary habits, enhancement of physical activates by exercising and behavioral changes(33). It is thought that these interventions can improve the anthropometric parameters of obesity by decreasing the BMI and waist circumference as well as improvement in hormonal body profiles such as reducing the insulin resistance and levels of serum testosterone and, increasing the levels of FSH with recovery of ovulatory functions and regular menstrual cycles(33).

First for the dietary modifications, researchers found that reducing the intake of carbohydrates with taking 30% of energy from a high protein diet may results in decreasing the insulin resistance compared to the traditional diet which consists of 15% of energy from proteins(33). It is also suggested that replacement of fatty foods with polyunsaturated fats or replacement of monounsaturated fats instead of polyunsaturated fats had no effects on serum androgens levels or lipid profiles in PCOs patients(33).

In addition, one study for caloric restriction conducted on 143 PCOs women with mean BMI of 35kg/m^2 with providing a diet consisting of 1000kcal/day for at least 7 months, results in losing 6.0kg, low androgens, reduction in abdominal fat and serum triglycerides(33). In general, a diet composed of 30% of fats, 50% of carbohydrates and 20% of proteins with more fibers is the most suitable diet for PCOs patients(33).

Second, a randomized controlled study included 90 PCOS women with exercising program for 40 minutes and for 3 days per week till 3 months resulted in decreasing the BMI and waist circumference compared to the control group with no exercise(33). There are two mechanisms suggested by several studies in which the exercise reduces the insulin resistance. First, it causes increasing of the metabolism in skeletal muscle cells thus it leads to improvement in insulin resistance(33). Second, it induces reduction of the visceral fat because it is highly active metabolically(33).

Also, the effects of exercise in restoring the ovulatory and reproductive functions is more prominent than the benefits of diet alone(33). Therefore, a combination of diet with moderately exercising for 30 minutes up to 5 days/week is more appropriate for obese women with PCOS(33).

Finally, several authors evaluated the beneficial effects of behavioral modification such as changing the abnormal eating habits and identifying good coping strategies. Furthermore, the doctors should offer rewards for patients who achieving weight loss and also providing a psychological support because losing weight decreases the rate occurrence of depression(33).

5.2 *Pharmacological therapies*

When lifestyle managements fail to improve the dyslipidemia, several pharmacological agents are proved by researches for reducing the obesity in PCOS patients such as the orlistat and statins(34). They found that the orlistat can inhabit the digestion of triglycerides by irreversible binding to pancreatic and gastric lipase enzymes as a result, it improves the metabolic or lipid profiles and leads to weight loss(34). Moreover, studies showed that statins inhabit the 3-hydroxy-3-methylglutaryl coenzyme A(HMG-CoA) reductase which is the rate limiting enzyme for cholesterol synthesis that results in lowering the lipid profiles such as the serum cholesterol, triglycerides and low density lipoprotein(LDL) and reducing the obesity in PCOS patients(35).

6. Prevalence of PCOS in Saudi Arabia

The incidence rate of PCOS within Saudi Arabian women is greatly increased as well as among the women around the world by affecting 5%-7% of their reproductive age(3). In a cohort retrospective study that conducted on 728 Saudi Arabian women under different criteria, they reported the prevalence of PCOS through the National Institutes of Health is estimated to be 8.7% in contrast to the prevalence of PCOs under the Rotterdam consensus criteria which is 11.9% and throughout the Androgen Excess Society criteria the prevalence is 10.2(3).

On the other hand, a cross sectional study performed in Almadinah Almunawwarah on 201 members aged 18-28 years they found 108 (53.7%) cases diagnosed with PCOS which is remarkably greater than the previous cohort study(3). The explanation for this result, is high occurrence of obesity within Saudi Arabian population reviewed by a study on 29-34 year old Saudi women diagnosed with PCOS with 64.5% of them are obese and 24.2% are overweight(3).

7. Obesity and PCOS

7.1 *Hyperinsulinemia*

Many women with PCOS are obese especially at the distribution of body fats in the abdominal area which is the cardinal feature for these patients(36). Some studies suggested that obesity plays a major role in pathogenesis of PCOS, while other studies showed that PCOS can contribute to development of obesity(36).

Obesity results in insulin resistance and as a compensatory response the body secretes large amount of insulin (Hyperinsulinemia) that leads to high amount of androgen production in the body(36). Insulin stimulates the ovaries to synthesize more androgen by binding to insulin-like growth factor-I receptors in the ovaries and also by stimulation of adrenocorticotropic hormone release from the anterior pituitary gland and androgen secretion from the adrenal cortex(36).

Furthermore, insulin could increase the granulosa cells sensitivity to follicle stimulating hormone resulting in development and growth of the antral follicles therefore, any conditions that lead to increase concentration of insulin in the general circulation, can predispose to hyperandrogenism and subsequent development of PCOS(36). Abdominal or central adiposity could also contribute to excess androgen from the adrenal glands or ovaries by several mechanisms independent to insulin(36).

7.2 *Leptin and cytokines*

Adipose tissue considered as endocrine organ by increasing or decreasing the secretion of some molecules for example, leptin and Adiponectin are adipokines in addition to interleukin 6 (IL-6) and tumor necrosis factor alpha (TNFa) which are cytokines (36).

Many researches demonstrated that levels of leptin hormone which suppress the appetite and reduce the weight of body are higher in women with PCOS compared to controls(37). Although higher levels of leptin in serum of PCOS patients, they do not respond to these levels by decreasing their food intake(37). This indicates the presence of leptin resistance due to restricted entry of leptin to the hypothalamus to acts on its receptors also, experiments on mice by producing mutations on leptin receptors results in insulin resistance(37).

Another adipokines is adiponectin that acts as anti-inflammatory hormone and has the ability to reduce the blood glucose levels by decreasing the gluconeogenesis and reinforces the action of insulin in the liver thus, researchers reported that adiponectin levels are much lower in obese PCOS patients because obesity reduces the expression of adiponectin receptors and inhabits its signaling which causes insulin resistance with elevated triglycerides levels(37).

(TNFa) is a cytokine that causes necrosis to the tumors originating from acute bacterial infections and its expression is higher in the adipose tissues which may leads to suppression of adiponectin production and increasing the insulin resistance along with inhibition of insulin signaling(38). Studies believe that levels of tumor necrosis factor alpha are elevated in women with PCOS compared to the healthy control group(38).

In addition, (IL-6) an inflammatory mediator is founded to be increased in serum of normal obese people and with PCOS patients(38). One study reported that interleukin 6 does not directly causes proliferation of theca interna cells instead, it causes upregulation of androgen receptors in the ovaries and might leads to insulin resistance(38). All these mechanisms explain how obesity promotes the development of PCOS(38).

On the other hands, evidences suggest that PCOS might leads to obesity because these women have excess androgen levels in their serum and are found to have great cravings for eating foods that rich in fats and carbohydrates as well as hyperinsulinemia and low basal metabolic rate can result in weight gain(39).

8. Anthropometric measurements of obesity in PCOS

Anthropometry is a simple, quantitative and noninvasive technique used for determining the size and fat composition of the body by measuring the body weight, high, skin fold thickness, BMI and circumference such as waist, hip or midarm(40). Several studies found that measuring the body mass index and waist circumference are the principle anthropometric parameters of obesity in PCOS women ,but other parameter such as waist to hip ratio (WHR) is also included(6).

Body mass index is calculated by dividing the weight in kilogram on the square meter of high (kg/m2)(6). BMI < 18.5 kg/m2 is considered underweight, BMI = 25.0-29.9 kg/m2 is overweight and BMI ≥ 30.0 kg/m2 is considered obese(41). In a cross sectional study that conducted at King Fahd National Guard Hospital in Saudi Arabia on total 62 women diagnosed with PCOS, they reported 7 patients (11.2%) at normal weight,15 patients (24.2%) are overweight and 40 patients (64.5%) are obese and their mean BMI is 31.91±6.40(42). Furthermore, they found a significant association between the numbers of ovarian cysts that measuring 2 to 9 mm with increasing in BMI values(42).

The reason for high values of BMI in PCOS women is that it has a positive correlation with the hormonal and metabolic body profiles such as the levels of serum TG, LDL, LH, FSH, testosterone and insulin hormones(43). A cross sectional study that performed at the King Fahd Hospital of Al- Dammam University on 50 PCOS women and 50 of non-PCOS women, showed the hormonal profiles are the follow: levels of LH are 8.9miu/ml, testosterone levels are 0.03nmol/l, insulin levels are15.3uU/dl which are significantly higher in the PCOS group than the healthy controls and the levels of FSH are 4.9miu/ml which are much lower in the cases than the controls **Table1**(43).

Table 1: Comparison of hormonal and lipid profiles between the cases and controls at the King Fahd Hospital of Al-Dammam University.

Hormonal and lipid profiles	Cases (n=50)	Controls (n=50)		P value
LH1	8.9miu/ml	6.03miu/ml		0.0026
FSH2	4.9miu/ml	6.6miu/ml		<0.0001
testosterone	0.03nmol/l	0.01nmol/l		<0.0001
Insulin	15.3uU/dl	11.9uU/dl		0.0025
Triglycerides	100.6mg/dl	80.6mg/dl		NS3
LDL4	103.7mg/dl	107.5mg/dl		NS3

Another prospective study performed on 180 PCOS patients and 20 healthy women at Ohud hospital, in AL-Madinah AL-Munawarah established that serum concentrations of triglycerides in these women are 129.8 mg/dl (P<0.001) and the concentrations of LDL are 115.2 mg/dl (P<0.01) which are significantly more elevated compared to the control group ,but the same study that was conducted at the King Fahd Hospital of Al- Dammam University found no significant difference in concentrations of serum TG and LDL between the cases and controls(43,44).

On the other hand, the waist circumference used for determining the amount fats present in the area of the abdomen and it can be measured by standing with the both arms at the sides then placing the tape measure at the midpoint between the top of iliac crest of the hip bones and the last rib that could be palpable(45). A cross sectional study on 90 Saudi PCOS women and 122 controls at the King Faisal Specialist Hospital reported that the waist circumference values in PCOS patients are 91.56cm which are significantly higher than the control group of 81.24cm (P<0.0001)(46).

The explanation for high waist circumference measurements in PCOS patients is because those women have increased levels of androgens, insulin and luteinizing hormone which lead to abdominal or central obesity and substantial increase in waist circumference(46).

Another measurement is the waist to hip ratio, in which the waist circumference divided on the widest point of the buttocks(45). The same study that performed at the King Faisal Specialist Hospital found that the waist to hip ratio in PCOS Saudi females is 0.858 which is significantly higher than the controls which is 0.759 (P<0.0001) . in addition, they suggested the same reasons that lead to increasing in waist circumference are also involved in higher waist to hip ratio(46).

Conclusion:-

PCOS is inherited autosomal dominant disorder seems to arise as a complex trait, that results from genetic mutations, insulin resistance, androgen excess and vitamin D deficiency but all researches agree about the presence of hyperandrogenism. It has many clinical features but the most common presenting picture is hirsutism. It is associated with long term complications involving irregular menstruations, type2 diabetes along with cardiovascular diseases and risk of cancer. There is a vicious cycle from which the central obesity initiates the pathogenesis of PCOS and that the PCOS alters the body functions and enhancement of abdominal obesity. Laboratory tests must search for hyperandrogenism and insulin resistance to investigate for PCOS and multiple ovarian cysts on ultrasound. Saudi Arabia has a high prevalence of PCOS due to increasing obesity and it is significantly correlated with high anthropometric values in BMI, waist circumference and waist to hip ratio. Exercising, dietary and behavioral changes in addition to orlistat and statins medications have been proved to be effective in weight loss.

[1] Luteinizing hormone

[2] Follicular stimulating hormone

[3] Not significant

[4] Low density lipoprotein

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