ISSN NO. 2320-5407

Journal homepage:http://www.journalijar.com
Journal DOI:10.21474/IJAR01

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH

RESEARCH ARTICLE

BIOCHEMICAL CHARACTERISTICSOF POLYCYSTIC OVARY SYNDROME.

Samir A.M. Zaahkouk, El-Yamany I. El-Zawahiri, Ahmed M. Bawdy, Fatma A. Eidand Rabea A. M. Mousa.

Zoology Department, Faculty of Science, Al-Azhar University, Cairo, Egypt

Manuscript Info

Manuscript History:

Received: 19 March 2016
Final Accepted: 26 April 2016
Published Online: May 2016

Key words:

Corresponding Author

Samir A.M. Zaahkouk,.

Abstract

The study carried out to evaluate the some Biochemical analysis that characterized of polycystic ovary syndrome that play role in the polycystic syndrome.

Martial and methods: - This study includes 20 woman attending Mansoura university woman Hospital, their age's ragas range from 20-35 years. This study was approved by the ethical and research committee of council of obstetrics and gynecology Mansoura university. Group I (study group): Consists of 10 women with polycystic ovary syndrome and Group II control group: Consist of 10 women.Eachcase-taking sample of fasting blood from veins to determine Biochemical analysis that characterized of polycystic ovary syndrome.

Results:- The weight , BMI ,ASAT , ALAT ,Cholesterol ,Triglyceride, LDL –Cholesterol, Fasting blood sugar ,Fasting insulin, Fasting blood sugar / Fasting insulin ratio, luteinizing hormone ,DAHESand AMHshowed significant increase changes when compared with control .while .non-significant in the Total protein , albumin globulin and albumin /globulin ratio , HDL –Cholesterol at the comparison with control group .On the other hand of the was FSH significant decrease in PCOS was at the comparison with control group .

Conclusion:-there are some Biochemical analysis that characterized of polycystic ovary syndromeas some hormones as FSH, LH, AMH, DAHEAS and insulin.

Introduction:-

Sayera *et al.* (2012) reported that, The PCOS is a heterogeneous condition that defined by the presence of two out of the following three criteria: Oligo- and/or anovulation; hyperandrogenism (clinically or biochemically) and polycystic ovary, with exclusion of other etiology. **Mei-J.*et al.* (2006)** reported that, **Polycystic** ovary syndrome (PCOS) is one of the most common endocrine disorders in women of reproductive age. At the Rotterdam revised consensus meeting in 2003, it was proposed that oligomenorrhea, clinical or biochemical hyperandrogenemia and the presence of polycystic ovaries should serve as the diagnostic criteria for PCOS **(Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group, 2004)** PCOS is increasingly recognized as a variant of the metabolic syndrome in women with the characteristic features of insulin resistance, central obesity, impaired glucose metabolism, dyslipidemia and hypertension . The increased risk of cardiovascular disease in women with PCOS, however, is still controversial. Women with PCOS have been reported to have lower serum high-density lipoprotein cholesterol (HDL-C) and higher serum triglyceride concentrations than those without PCOS. Low HDL-C has been reported to be the most important lipoprotein profile predictor for the occurrence and mortality of cardiovascular disease.

Praveen *et al.* (2016) resulted that, polycystic ovarian syndrome (PCOS) is a complex and multifactorial disorder believed to be the consequence of a complex interaction between genetic, immunological, and environmental factors

Khayyat*et al.* (2012) recorded the height, weight of every patient was measured and recorded on their first visit, and BMI was calculated. Patients were grouped according to their BMI into three groups of normal with BMI of 20 to 24.9, overweight (25 to 29.9) and obese (≥30). Increase body mass index (BMI) with clinical symptoms in polycystic ovary syndrome (PCOS) women. The findings of this study indicated that the overweight/obese women with PCOS are at an increased risk for sonographic view of polycystic ovaries. **(Seddigheh *et al.* 2015)**

Gangale *et al*. (2011) reported that, case-control study from Chile showed a statistically significant difference in elevated ALT levels between 41 PCOS patients compared to 31 age- and body mass index (BMI)- matched healthy women (39% vs 3.1%, respectively), using a cut-off > 25 U/L, according to normal values for healthy Chilean women.**Gangale *et al.* (2011)** reported that, case-control study from Chile showed a statistically significant difference in elevated ALT levels between 41 PCOS patients compared to 31 age- and body mass index (BMI)-matched healthy women (39% vs 3.1%, respectively), using a cut-off > 25 U/L, according to normal values for healthy Chilean women.

Banaszewska *et al.* (2003) resulted LH/FSH ratio greater than 2 was accepted as abnormal, and it was found in 54 women (45.4%; I group). Normal gonadotropin ratio was detected in 65 women (55%; group II). Statistically significant differences were noted between groups with normal and elevated LH/FSH ratio in the following parameters: BMI (body mass index), serum insulin, and LH levels. Further analysis revealed that the majority of women with elevated insulin concentrations belong to the group with normal LH/FSH ratio.

Sunita et al. (2012) resulted that, Hormonal profile of PCOS was studied in 102 Indian women. Serum levels of Luteinizing hormone (LH), Follicle stimulating hormone (FSH), LH: FSH ratio, Prolactin (PRL), Thyroid-stimulating hormone (TSH), Dehydroepiandrosterone (DHEA), Testosterone, fasting blood glucose (FBG), fasting insulin levels and Homeostasis Model Assessment (HOMA) value were estimated. The mean LH and FSH levels are 12.54 ± 5.87 and 5.70 ± 1.80 (IU/L) respectively. The mean LH: FSH ratio is reversed and is more than two (2.23±0.94). Mean PRL, TSH, and testosterone levels show normal ranges. Mean fasting insulin (16.27±13.27 µU/ml) and HOMA (3.509±2.621) are high with 79.31% prevalence of insulin resistance. In all the patients, both LH and FSH are positively correlated with testosterone. In normal weight patients, PRL and LH: FSH are positively correlated. In overweight/obese serum, LH and DHEA are positively correlated. A positive correlation was observed between testosterone and PRL in overweight/obese. On sub-grouping data of gonadotropin levels with respect to different days of menstrual cycle, LH levels and LH: FSH ratio but not FSH levels show significant intergroup variation. The authors conclude that, low levels of FSH is persistent irrespective of day and phases of menstrual cycle, the reversal of LH: FSH is mainly because of lower FSH and the physiological cyclical pattern of gonadotropin altered in PCOS with partial preservation of the cyclical variation of LH but not of FSH. Insulin resistance is independent of BMI and is common in Indian PCOS women.

Nida (2014) concluded that, Anti-mullerian Hormone (AMH) is a member of the transforming growth factor β family of growth and differentiation factors. It has an integral role in the intrauterine development and sex differentiation of the male fetus. It is secreted from the Sertoli cells of the developing testes inhibiting ipsilateral mullerian duct development and thereby allowing the Wolffian duct system to prevail. However, the role of AMH across the female reproductive life span has only more recently become known. In the ovary, AMH has an inhibitory effect on primordial follicle recruitment as well as on the responsiveness of growing follicles to Follicle-Stimulating Hormone (FSH). The ovary-specific expression pattern in granulosa cells of growing non-selected follicles makes AMH an ideal marker for the size of the ovarian follicle pool and a prognostic factor for fertility potential.

Chang *et al.* (2005) reported that, the adrenal cortex synthesizes all the three major androgens; dehydroepiandrosterone sulfate (DHEAS), androstenedione and testosterone, and this is the other major site of female androgen production, besides the ovaries. DHEAS is almost exclusively (97-99%) produced by the adrenal cortex and androstenedione is produced in both the adrenal gland and the ovaries , whereas 25% of testosterone is synthesized by the adrenal gland, 25% in the ovary and the remaining part being produced through peripheral conversion from androstenedione in liver, adipose tissue and skin . Around 60-80% of PCOS women have high concentrations of circulating testosterone. In PCOS women, the prevalence of DHEAS excess is 20-30%, depending on ethnicity and DHEAS levels decline up to the age of ~ 45 years. The increased DHEAS levels in PCOS women

compared with controls is verified up to the perimenopausal ages. However, the mechanisms of the adrenal androgen excess in PCOS is still unclear, although it has been proposed that it may result from increased metabolism of cortisol, which could lead to decreased negative feedback on ACTH secretion.

Moran *et al.* (1999) concluded that, Clinically, the measurement of circulating levels of the AA metabolite DHEA sulfate (DHEAS) has been traditionally used as a marker for AA excess because this steroid is 97–99% of adrenocortical origin, the second most abundant steroid after cortisol (F), relatively stable throughout the day and the menstrual cycle, because of its relatively long half-life, and easily measured. Excess AA levels, particularly elevations in the levels of the dehydroepiandrosterone (DHEA) metabolite DHEAS and 11-hydroxyandrostenedione (11OHA4), were initially reported in 40–60% of patients with PCOS. However, in most of these early studies criteria for the selection of PCOS patients were different from those currently used. Moreover, it is clear that several factors, including age and race, should be considered when estimating the prevalence of AA excess in PCOS, because AAs begin to decline after the age of 30 years in both normal women and women with PCOS.

Carmina *et al.* (1992) resulted that, in a retrospective study of 145 hyperandrogenic patients, we found that hyperandrogenic patients with high DHEAS levels were younger, in addition to being thinner and more hirsute, than hyperandrogenic women with lower DHEAS levels. The impact of race on the prevalence of AA excess in PCOS is unclear. In one report the prevalence of AA excess among PCOS patients was found to be similar among Italian, US, Hispanic-American, and Japanese women. However, only small groups of patients compared. To reevaluate the prevalence of AA excess in PCOS taking into account race and age-related changes in AAs, we undertook a study of 213 (27 black and 186 white) women with PCOS and 182 (88 black and 94 white) age-matched healthy eumenorrheic nonhirsute women (controls) .

Huerta*et al.* (1999) concluded that, the diagnosis of PCOS based on hyperandrogenism and chronic anovulation, consistent with the National Institutes of Health 1990 criteria. DHEAS levels were significantly lower in black than white controls, whereas fasting insulin and body mass index (BMI) were higher in black controls, and DHEAS levels decreased similarly with age in control and PCOS women of either race. Body mass and fasting insulin had little impact on circulating DHEAS levels in healthy women. Among PCOS patients, these parameters were negatively associated with circulating DHEAS levels among white, but not black patients. For each race and age group, the upper 95% normative values for log DHEAS was calculated, and the number of PCOS subjects with log DHEAS values above this level assessed. The prevalence of supranormal DHEAS levels was 33 and 20% among black and white women with PCOS, respectively—not a significant difference.

Sanchez *et al.* (2002).resulted that, these data indicate that AA excess, defined by the circulating level of DHEAS, is somewhat less common in PCOS than previously reported, affecting between 20 and 30% of affected women when using age- and race-adjusted normative values. However, it also appears that women with absolute DHEAS excess simply represent the upper edge of the normal DHEAS distribution in the general population, not a separate population. For example, cluster analysis failed to reveal any specific subpopulations of DHEAS levels among our patients with PCOS. Finally, there may be significant differences in mean DHEAS levels between white and black control women.

Mehmet *et al.* (2015) resulted that, finally, we should note that DHEAS, does not uniformly reflect AA secretion in response to adrenocorticotropic hormone (ACTH) in normal or hyperandrogenic patients. For example, only 50% of patients with 21-OH-deficient NCAH have a supranormal DHEAS levels. Witness also the profound suppression in DHEAS levels that occurs in NCAH patients treated with glucocorticoids despite the still elevated production of low-dose A4. Likewise, note should be taken of the increase in DHEAS in response to exogenous testosterone administration to oophorectomies women, despite the absence of any change in the AA response to acute ACTH stimulation. Consequently, it is apparent that a number of factors may alter DHEAS levels without modifying adrenocortical AA production, most likely through regulation of DHEA sulfotransferase (DHEA-ST) activity. Hence, the investigation of those mechanisms underlying the AA excess of PCOS requires evaluation not only of DHEAS levels, but also of adrenocortical biosynthesis (usually measurable by the response to acute ACTH stimulation). In our study, increased BMI observed with a correlation between DHEAS levels and a similar relationship because of work done by Park and colleagues also found.

Material and methods:-

This study includes 20 woman attending Mansoura university woman Hospital, their age's ragas range from 20-35 years. This study was approved by the ethical and research committee of council of obstetrics and gynecology Mansoura university.The 20 women are divided into two groups

Group I control group:-

Consist of 10 women attending in family planning clinic seekingContraception.Mean age of the control group was **25.4 ± 4.57** year while the mean BMIwas**26.4 ± 2.22** kg/m^2.

Group II (study group):-

Consists of 10 women with polycystic ovary syndrome diagnosed According to Rotterdam criteria 2004 in which to diagnose PCOS two from**1**. Oligo and /or anovulation.**2.** Clinical and/ or biochemical features of hyperandrogenism for example acne and Hirsutism**3**. The presence of polycystic ovary morphology by U/S. (**Rotterdam ESHRE/ESRM sponsored PCOS consensus workshop group 2004**) Mean patients age was **27.1 ± 5.66** and the mean BMI was **29.99 ± 2.86** Kg/m^2.

Inclusion criteria1 age 20-35 years old and healthy two women attending gynecology outpatient clinic of El-Mansoura University hospital**.**

Exclusion criteria1-women with amenorrhea.**2**-Hormonal treatment during the three months before the study e.g. Induction of ovulation.**3**-history of ovarian operations including drilling. **4**-Associated medical problems e.g. diabetes.**5**-Associated endometriosis if previously diagnosed.6. The presence of pelvic pathology.

History:-

History:-Each was questioned about her age, duration of marriage. Duration of infertility, and her full menstrual history. Symptoms suggestive of Endocrinological disorders in the form hirsutism. Galactorrhea, thyroid Dysfunction end diabetes.A full medical and surgical history was taken.

The serum cholesterol (mg/dl) was estimated by enzymatic colorimetric (CHOD- PAP) method, according to **Meiattini*et al.* (1978)**, using kit, supplied by Spinreact, S. A. Spain. The serum triglycerides (mg/dl) was estimated by enzymatic colorimetric (GPO- PAP) method, according to **Bucolo and David (1973)** using kit, supplied by Spinreact, S. A. Spain. Serum HDL cholesterol (mg/dl) was estimated by enzymatic –colorimetric (GOD PAP) method, according to**Burtiset al.(1999)** using kit, code 1001095 supplied by Spinreact, S. A. Spain. Determination of low-density lipoprotein-cholesterol (LDL-C) level (mg/dl): The LDL-C calculations were conducted according to the formula of **Wieland and Seidel (1982).**

LDL-C = Total cholesterol – (TG/5) – HDL-C

The serum glucose was estimated by enzymatic –colorimetric (GOD PAP) method, according to Burtis *et al*.(1999) using kit, supplied by Spinreact, S. A. Spain. The serum ASAT and ALAT was estimated by Liqui UV test method, according to **Schumann and Klauke, (2003)** using kit, supplied by Human, Germany. The serum total protein was estimated by Liquicolor (photometric colorimetric) test, according to **Young (2001),** using kit, supplied by Human, Germany. The serum Albumin was estimated by Liquicolor (photometric colorimetric) test BCG method, according to **Johnson *et al.* (1999)** using kit, supplied by Human, Germany. (gldl). Serum anti-Mullerian hormone was measured using The DSL Mullerian inhibiting substance/ anti-Mullerian hormone (MIS/AMH) enzyme- linked lmmunosorbent (ELISA) Kit. According to**Gruijters *et al.* (2003).** Diagnostic systems laboratories. Inc. Webster. Texas. USA.Direct immunoenzymatic determination of insulin level in human serum or plasma by Dimetra kits according to **Gerbitz, (1980)**.Direct immunoenzymatic determination of the Follicle-Stimulating Hormone (FSH) in human serum or plasma. According to **Gerbitz, (1980).**Direct immunoenzymatic determination of the luteinizing hormone (LH) in human serum or plasma (mIU/ml) according to**Lenton *et al.* (1982).** Determination of Testosterone by enzyme immunoassay in human serumby**Ekins(1990**) this kit is for in vitro Diagnostic use. By ALBCO kits, USA.The DRG DHEA-S ELISA is an enzyme immunoassay for the quantitative in vitro diagnostic measurement of DHEA-S in serum and plasma by**Labrie *et al.*, (2005).**

Results:-

In the present investigation of the Anthropometric of polycystic ovary syndrome (PCOS) women and controls showed in table (1) showed non-significant in increase ($p>0.05$) in the age and high (27.1 ± 5.66) and (1.71 ± 0.044) at the comparison with control group (25.4 ± 4.57) and (1.73 ± 0.047). The weight and BMI showed significant increase changes ($p<0.05$) (90.6 ± 9.5) and (29.99 ± 2.86) when compared with control (77.7 ± 9.2) and (26.4 ± 2.22). Data resulted in table (1) and illustrated in figures (1) showed highly significant in increase ($p<0.01$) in the ASAT and ALAT and high increase (28.5 ± 0.93) and (33.5 ± 4.12) at the comparison with control group (20.6 ± 1.4) and (19.6 ± 4.55) respectively. Obtained data in table (1) and illustrated in figures (2) showed non- significant ($p>0.05$) in the Total protein , albumin globulin and albumin /globulin ratio (7.113 ± 0.305), (4.40 ± 0.38), (2.71 ± 0.457) and (1.68 ± 0.45) at the comparison with control group (6.90 ± 0.265), (4.40 ± 0.38), (2.70 ± 0.24) and (1.60 ± 0.20) respectively. Data recorded in table (1) and illustrated in figures (3) showed significant in increase ($p<0.05$) in the Cholesterol and Triglyceride and increase (196.1 ± 18.34) and (93.5 ± 22.8) at the comparison with control group (168 ± 23.8) and (69.3 ± 16.7) respectively. On the other hand of the HDL –Cholesterol was non-significant increases ($p>0.05$) where the PCOS was (54.7 ± 6.72) at the comparison with control group (49.7 ± 7.07) and significant in increase ($p<0.05$) in the. LDL –Cholesterol and increase (122.71 ± 11.2) at the comparison with control group (104.44 ± 19.58).Data resulted in table (1) and illustrated in figures (4) showed highly significant in increase ($p<0.01$) in the Fasting blood glucose and Fasting insulin and high increase (90.10 ± 6.607) and (9.41 ± 2.47) at the comparison with control group (79.4 ± 8.488) and (6.33 ± 2.32) respectively. Obtained data in table (1) showed high significant ($p<0.01$) in the Serum fasting blood glucose / serum fasting insulin ratio (10.14 ± 2.50) at the comparison with control group (13.82 ± 4.07). Data resulted in table (1) and illustrated in figures (6) showed highly significant decrease ($p<0.01$) in the follicle stimulating hormone (5.156 ± 0.615) at the comparison with control group (7.485 ± 2.058). Obtained data in table (1) showed highly significant in increase ($p<0.01$) inluteinizing hormone (11.39 ± 2.57) at the comparison with control group (3.58 ± 0.94). Obtained data in table (1) and illustrated in figures (7) showed high significant increase ($p<0.01$) in the dehydroepiandrosterone sulfate (2327.9 ± 337.18) at the comparison with control group (1911.7 ± 227.98). While the AMH showed high significant increase ($p<0.01$) (75.41 ± 15.2) at the comparison with control group (49.335 ± 9.592). The total testosterone showed high significant increase ($p<0.01$) (0.630 ± 0.13) at the comparison with control group (0.278 ± 0.129. Obtained data in table (1) and illustrated in figures (8&9) showed high significant increase ($p<0.01$) in the dehydroepiandrosterone sulfate (2327.9 ± 337.18) at the comparison with control group (1911.7 ± 227.98). While the AMH showed high significant increase ($p<0.01$) (75.41 ± 15.2) at the comparison with control group (49.335 ± 9.592). The total testosterone showed high significant increase ($p<0.01$) (0.630 ± 0.13) at the comparison with control group (0.278 ± 0.129).

Results:-

cases	Normal	PCOS	P value
Age (year)	25.4 ± 4.57	27.1 ± 5.66^{ns}	0.529
Weight (kg)	77.7 ± 9.2	90.6 ± 9.5*	0.014
Height (meter)	1.73 ± 0.047	1.71 ± 0.044^{ns}	0.99
BMI (kg/m^2)	26.4 ± 2.22	29.99 ± 2.86*	0.013
SGOT (ASAT) (U/L)	20.6 ± 1.4	28.5 ± 0.93**	0.001
SGPT (ALAT) (U/L)	19.6 ± 4.55	33.5 ± 4.12***	0.000
ALAT/ASAT Ratio	0.97 ± 0.22	1.21 ± 0.2*	0.016
Total protein (gm./dl)	6.90 ± 0.265	7.113 ± 0.305ns	0.180
Albumin (gm./dl)	4.23 ± 0.24	4.40 ± 0.38ns	0.254
Globulin (gm./dl)	2.70 ± 0.24	2.71 ± 0.457ns	0.254
Albumin/globulin ratio	1.60 ± 0.20	1.68 ± 0.45ns	0.634
Cholesterol (mgldl)	168 ± 23.8	196.1 ± 18.34*	0.028
Triglyceride (mgldl)	69.3 ± 16.7	93.5 ± 22.8*	0.041
HDL –Cholesterol (mgldl)	49.7 ± 7.07	54.7 ± 6.72^{ns}	0.093
LDL –Cholesterol (mgldl)	104.44 ± 19.58	122.71 ± 11.2*	0.035
Fasting blood glucose (mgldl)	79.4 ± 8.488	90.10 ± 6.607**	0.001
Fasting insulin (uIU/ml)	6.33 ± 2.32	9.41 ± 2.47**	0.006
FBS/ insulin (fasting) ratio	13.82 ± 4.07	10.14 ± 2.50**	0.009
FSH mIU/ml	7.485 ± 2.058	5.156 ± 0.615**	0.001
LH mIU/ml	3.58 ± 0.94	11.39 ± 2.57**	0.001
LH/FSH	0.4837 ± 0.06	2.21 ± 0.45***	0.000
DHEAS ng/ml	1911.7 ± 227.98	2327.9 ± 337.18**	0.008
AMH Pmol/liter	49.335 ± 9.592	75.41 ± 15.2**	0.001
Total testosterone ng/ml	0.278 ± 0.129	0.630 ± 0.13***	0.000

Mean with dissimilar superscript letter are significantly different at (P<0.05) (p<0.05) =* (p<0.01) =** (p<0.001) =***LH: luteinizing hormone; FSH: follicle stimulating hormone; total T: total testosterone; DHEAS: dehydroepiandrosterone sulfate. AMH: Anti Mullerian hormone.

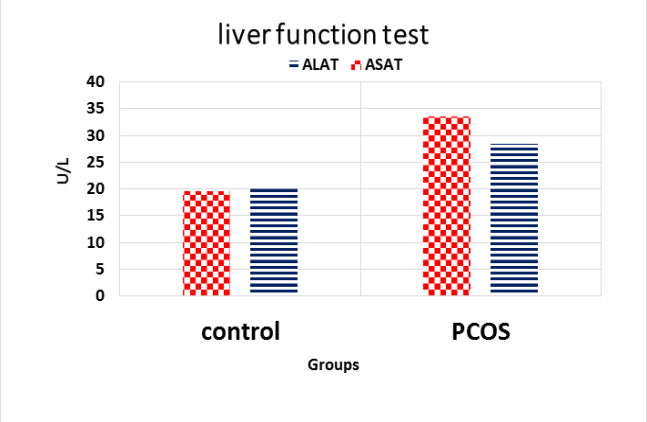

Fig. (1): Serum Aspartate Aminotranferase and Alanine Aminotranferase of polycystic ovary syndrome (PCOS) women and controls.

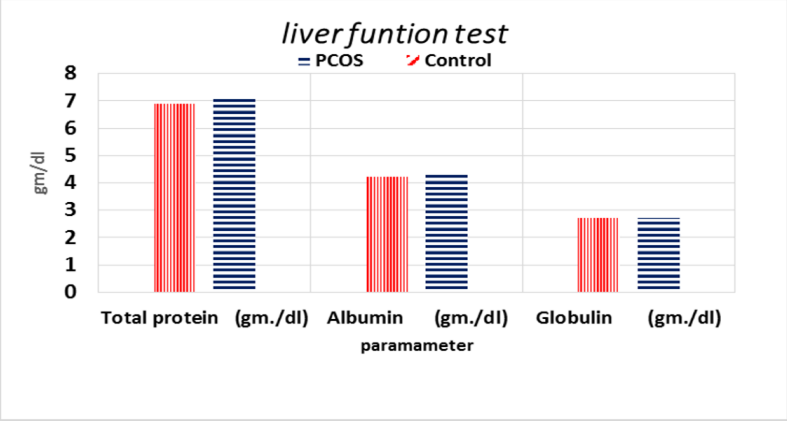

Figure (2): serum total protein, albumin Figure (2): serum total protein, albumin & globulin of polycystic ovary syndrome (PCOS) women and controls

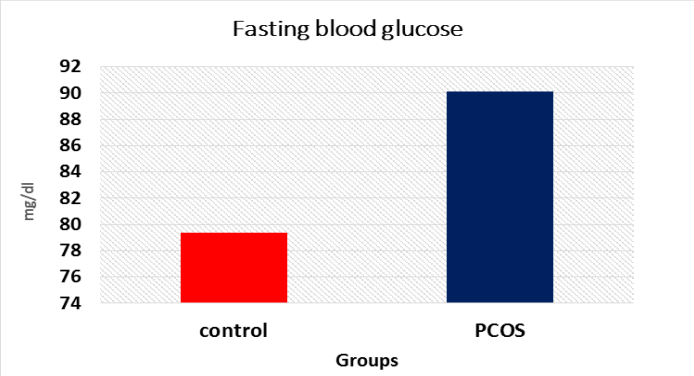

Figure (3): Fasting blood glucose of polycystic ovary syndrome (PCOS) women and controls.

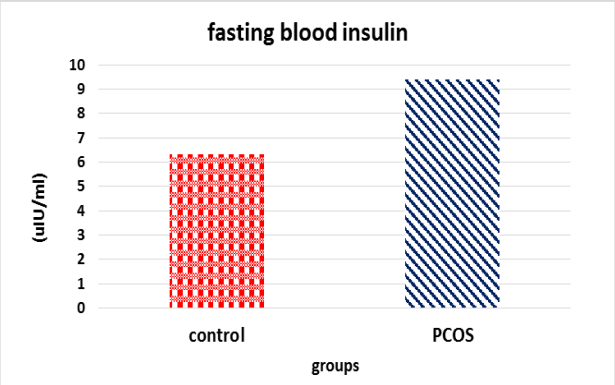

Figure (4): Fasting insulin of polycystic ovary syndrome (PCOS) women and controls.

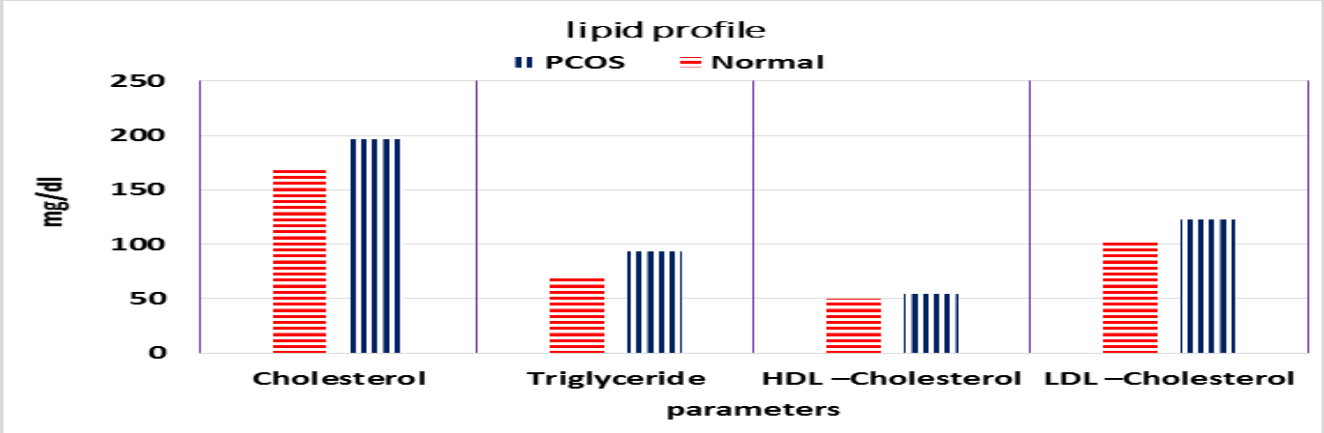

Figure (5): Lipid profile of polycystic ovary syndrome (PCOS) women and controls

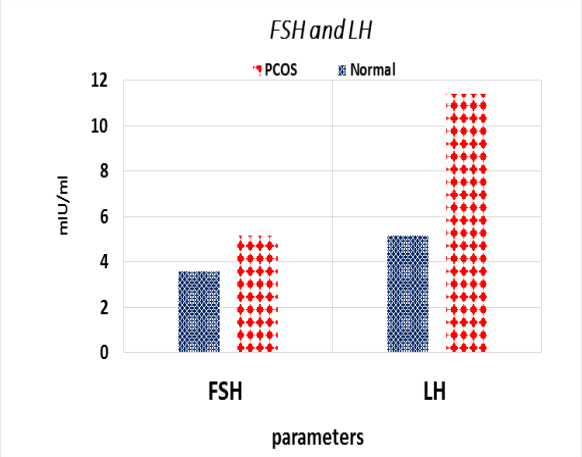

Fig. (6): LH: luteinizing hormone and FSH: follicle-stimulating hormone

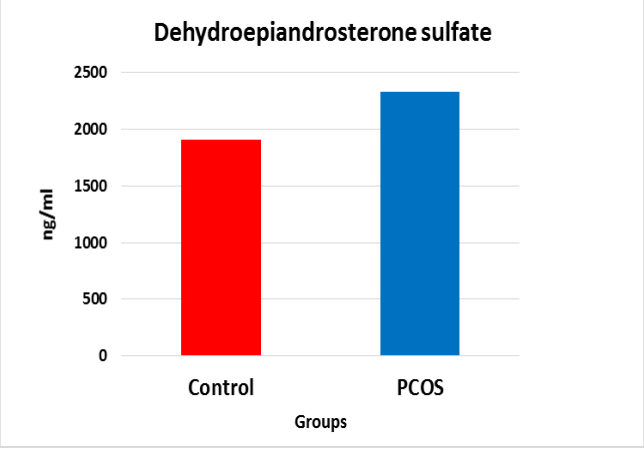

Fig. (7): DHEAS: dehydroepiandrosterone sulfate of polycystic ovary syndrome (PCOS) women and control.

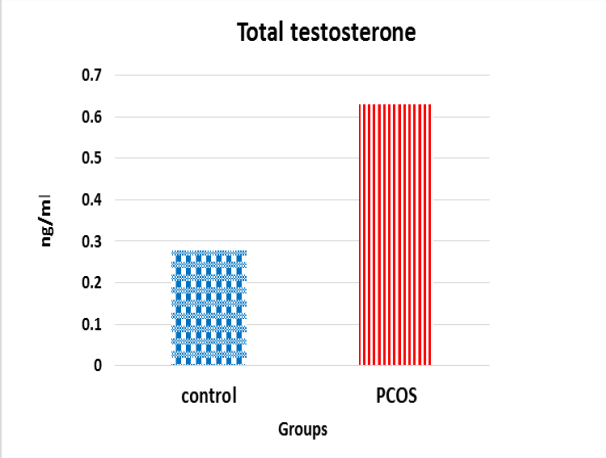

Fig. (8): Total testosterone of polycystic ovary syndrome (PCOS) Women and control.

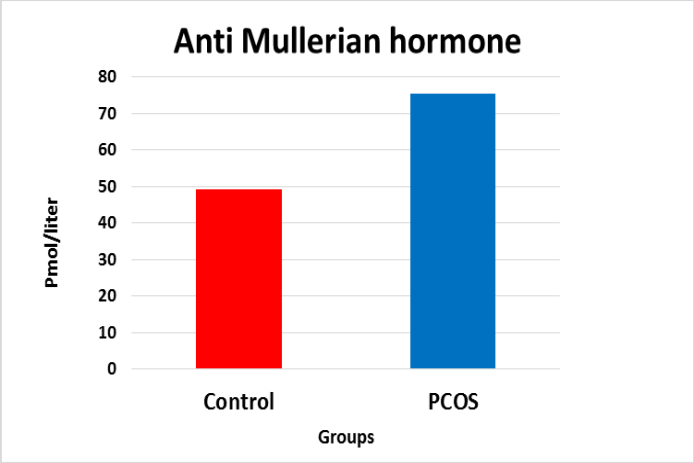

Fig. (9): AMH of polycystic ovary syndrome (PCOS) Women and control.

Discussion:-

The first observation from the physical examination increase the body mass index and Wight in polycystic ovary syndrome woman when compared with normal woman. This result correlate with previous studies that recorded increase body mass index (BMI) with clinical symptoms in polycystic ovary syndrome (PCOS) women. The findings of this study indicated that the overweight/obese women with PCOS are at an increased risk for sonographic view of polycystic ovaries. **Seddigheh et al. (2015)**.another study showed increase of weight and BMI of PCOS when compared with control. **Khayyat et al. (2012)**. Increase BMI due to Insulin resistance is strongly associated with androgenic type of obesity (abdominal). (**Azziz et al. 1998)**

In addition, **Evangeline (2014)** showed that elevated BMI in PCOS when compared with control. In this study concluded the increase of cholesterol, LDL-Cholesterol and triglyceride in PCOS when compared with control while increase HDL-Cholesterol non-significant these studies agree with **Setji*et al*. (2006)** showed that increase of triglycerides. Another study show total cholesterol and LDL-cholesterol were positively associated only with the presence of PCOS while No association was observed between HDL-cholesterol levels and the presence of PCOS.**Cristian*et al.*(2012)**.However, this study disagree with **Cristian *et al.*(2012)** that showed No difference was observed between groups in terms of triglycerides levels. Dyslipidemia, including elevations in circulating LDL - cholesterol, the precursor to sex steroid biosynthesis, is common in women with PCO s. Statins have multiple actions that include inhibition of the enzyme hydroxymethylglutaryl coenzyme a reductase, which leads to decreased production of cholesterol (thus reducing circulating concentrations of cholesterol). In addition, there is some evidence that ovarian T production may be reduced by administration of statins (**Legro*et al.* 2007).** This effect may be due, at least in part, to inhibition of theca cell growth and by decreasing the concentration of precursor for production of androstenedione (**Balen*et al.* 2003**). Furthermore, statins appear to have antioxidant properties. Clinical trials of statins alone or in combination with other medications among women with PCOs are limited in number, and conclusive evidence that statins ameliorate PCOs symptoms is lacking, although improvements in hyperandrogenemia have been noted (**Mikola*et al.* 2001**). Further recent data show that statin use may increase the risk for developing T2 DM (**Haakova*et al.* 2003**).while triglyceride increase duo to fats that provide energy for the cell. Like cholesterol, they are delivered to the body's cells by lipoproteins in the blood. A diet with a lot of saturated fats or carbohydrates will raise the triglyceride levels, liver dysfunction resulting from hepatitis, extra hepatic biliary obstruction or cirrhosis, diabetes mellitus is associated with the increase.(**Young2001**).They also demonstrate an increase in LDL particle number and a borderline decrease in LDL size and suggest that androgens may play a more significant role in pathogenesis of lipid abnormalities in PCOS (**Sidhwani*et al.* 2001**). The mechanism by which hyperandrogenism may contribute to development of lipid abnormalities in PCOS is not clear. Hyperandrogenism may lead to the abnormalities in lipoprotein profile by working directly at the liver, or it may alter body composition by favoring central adiposity. (**Echiburú *et al.* 2012)**The obtained results of this showed increase of aspartate aminotransferase and alanine aminotransferase compared with women of PCOS with normal women. While not significant increase in total protein, serum albumin

and globulin. These results agree with **Setji*et al.* (2006)** that recorded elevated in recorded Fifteen percent (29 of 200) had aspartate aminotransferase and/or alanine aminotransferase more than 60 U/liter. Also, **Gangale *et al.* (2011)** reported that, case-control study from Chile showed a statistically significant difference in elevated ALT levels between 41 PCOS patients compared to 31 age- and body mass index (BMI)- matched healthy women (39% vs 3.1%, respectively), using a cut-off > 25 U/L, according to normal values for healthy Chilean women. Moreover, **Barfield *et al.* (2009)** Reported that, elevated ALT and AST levels had elevated aminotransferase levels.AST is widely distributed with high concentrations in the heart, liver, skeletal muscle, kidney and erythrocytes. Damage or disease to any of these tissues such as myocardial infarction, viral hepatitis, liver necrosis, cirrhosis and muscular dystrophy may result in raised serum levels of AST. (**Zilva*et al.*1979**)In addition**, Vassilatou *et al.* (2010)** reported that, case-control study from Greece showed a significant difference of ALT and AST PCOS patients compared to healthy women using a cut-off > 40 U/L. All patients and controls with metabolic syndrome had HS. Duo to The ALT is a cellular enzyme, found in highest concentration in liver and kidney. High levels are observed in hepatic disease like hepatitis, diseases of muscles and traumatisms, its better application is in the diagnosis of the diseases of the liver. When they are used in conjunction with AST aid in the diagnosis of infarcts in the myocardium, since the value of the ALT stays within the normal limits in the presence of elevated levels of AST.(**Young 2001)**

On the other hand, the present study indicated In this study showed highly significant in increase in the Fasting blood sugar and Fasting insulin and high increase at the comparison with control group and showed high significant in the Fasting blood sugar / Fasting insulin ratio at the comparison with control group this results agree with **Setjiet al. (2006)** showed increase fasting glucose and fasting insulin in PCOS when compared with normal woman**.** Also showed high significant ($p<0.01$) in the Fasting blood glucose/ Fasting insulin ratio at the comparison with control group .**Joselyn *et al.* (2014)** Moreover, **Setji*et al.* (2006)** recorded increase the fasting insulin in PCOS when compared with control. Increase the insulin due to several conditions in which insulin disturbance is pathologic: diabetes mellitus, insulinoma, metabolic syndrome and polycystic ovary syndrome. There are two types of diabetes mellitus: type 1 (autoimmune-mediated destruction of insulin producing beta cells in the pancreas resulting in absolute insulin deficiency), and type 2 (multifactor syndrome with combined influence of genetic susceptibility and influence of environmental factors, the best known being obesity, age, and physical inactivity, resulting in insulin resistance in cells requiring insulin for glucose absorption. This form of diabetes is strongly inherited).(**Gerbitz 1980)**.

In the present studies Showed highly significant decrease in the follicle stimulating hormone at the comparison with control group. While highly significant in increase in luteinizing hormone at the comparison with control group. On the other hand high significant increase the dehydroepiandrosterone sulfate at the comparison with control group. While the AMH showed high significant increase at the comparison with control group. The total testosterone showed high significant increase at the comparison with control group. This result agree with **Muhammad and Nabila (2015)** resulted the elevated LH in PCOS has also been reported earlier.12 However, the elevation in LH level, in this study, was significantly intense than the reported values. The LH: FSH ratio in PCOS group, in comparison to control subjects, was raised by 18-folds in this study. Interestingly, PCOS women with hyperinsulinemia and overproduction of LH had significantly higher serum levels of dehydroepiandrosterone sulphate. In the remaining groups, DHEAS concentration was normal. It is still not fully under-stood how insulin influences the adrenal androgen secretion. The negative correlation between insulin levels and dehydroepiandrosterone sulphate production have been found. On the other hand, there are also studies that do not confirm correlation between insulin activity and adrenal androgen production **(Azziz et al., 1998).**

The elevated LH due to Nowadays it is believed that elevated LH level occurs more rarely in a group of patients with insulin resistance and hyperinsulinemia, than in-group without hyperinsulinemia. This observation was confirmed in a presented group of women, in which normal gonadotropin ratio 1:1 was observed in up to 72% of patients with hyperinsulinemia. One may speculate that additionally to, that is considered to be a strongest androgen production stimulator, in women with normal LH level additional stimulators of steroidogenesis exist. Most probably it is insulin and IGF-I. Thus, it could have been expected that the most severe clinical symptoms and greater androgen concentration would appear in women with hyperinsulinemia and overproduction of LH. However, the mean testosterone levels in the studied women were independent of insulin and LH concentrations. Hirsutism of greater sever it was observed in a group of women with hyperinsulinemia and LH/FSH ratio > 2 when compared with women with hyperinsulinemia and normal gonadotropin ratio **(Banaszewska*et al.*2003)**. Interestingly, PCOS women with hyperinsulinemia and overproduction of LH had significantly higher serum levels of

dehydroepiandrosterone sulphate. In the remaining groups, DHEAS concentration was normal. It is still not fully understood how insulin influences the adrenal androgen secretion. The negative correlation between insulin levels and dehydroepiandrosterone sulphate production have been found. On the other hand, there are also studies that do not confirm correlation between insulin activity and adrenal androgen production **(Azziz et al., 1998).**

In the present study concluded elevated serum testosterone in polycystic ovary syndrome Serum testosterone level so is the best marker for ovarian hyperandrogenism, and dehydroepiandrosteronesulfate is the best adrenal marker this results agree with **Carmina*et al.*(1992)** concluded that, The measurement of free testosterone provides a higher diagnostic yield for ovarian hyperandrogenism because levels of sex-hormone binding globulin are decreased. However, clinical assays used to test this measure vary considerably, affecting its reliability. It is important to point out that hyperandrogenemia is not synonymous with hirsutism or acne. Some ethnic groups (for example, Asians) have substantialhyperandrogenism (elevated levels of testosterone and dehydroepiandrosterone sulfate) without any significant skin manifestations.

In the present study, we used diagnostic criteria recommended by **Carmina *et al.(*1992**) for selecting the patients. It has been suggested that serum AMH levels were increased in PCOS .Although AMH seems a promising diagnostic tool, Hart et al failed to demonstrate serum AMH was a reliable predictor of polycystic ovarian morphology or for the presence of PCOS in the general adolescent population (**Hart *et al.*2010**). Routine serum testing for AMH levels for the diagnosis of PCOS is controversial. In the present study, AMH levels among study groups were different. PCOS subjects had significantly higher AMH levels duo to the positive correlation between AMH and ovarian volume. AMH also positively correlated with total testosterone. We found AMH levels and testosterone high in adolescent PCOS group. In previous studies, AMH levels were found to be associated significantly with testosterone especially in adolescents. This finding is harmonious with our results. We think that AMH can be a useful marker to determine the adolescent population who have a tendency to develop PCOS. Further studies are required with larger sample sizes. We also think that subgroups in adolescent PCOS group should be determined which are associated more significantly with elevated AMH levels in subsequent studies. Moreover, AMH levels in PCOS women were found to be approximately three- fold higher that those of healthy fertile control. Although the increase in AM H levels in PCOS women was thought to be due to the increase in small antral follicles, a recent stud y showed that AMH production is 75 times higher per granulosa cell in PCOS patients than in granulosa cells innormal ovaries**.(Pellatt*et al.* 2007).**Furthermore, AMH concentrations in the follicular fluid were five times higher in the follicles of womenwith anovulatory PCOS compare d with women who were ovulatory**(Das*et al.* 2008)**In this study, the rate of age-related decline inAMH concentrations was found to be smaller in PCOSpatients compared with healthy fertile patient s. Whether or not the aforementioned finding is due to a slower depletion of thefollicular pool in PCOS patients requires further rese arch. The sensitivity and specificity of AMH-base d detect ion of PCOS in Taiwanese women aged 29e 38 years were calculated to be74% and 79%, respectively, using an AMH cut- off value of3.5 ng/mL. Whether it is helpful or not to use AMH screening tool for PCOS require s future investigations.(**Seifer*et al.* 2009).**

References:-

1. **Azziz, R.; Black, V.; Hines, G.; Fox, L. and Boots L. (1998):** "Adrenal androgen excess in the polycystic ovary syndrome: sensivity and responsitivity of the hypothalamic-pituitary-adrenal axis." J ClinEndocrinolMetab, 1998; 83: 2317-23.
2. **Balen, A.; Laven, J.; Tan, S. and Dewailly, D. (2003):"** Ultrasound assessment of the polycystic ovary: international consensus definitions." Hum Reprod Update. 9:505–514.
3. **Banaszewska, B.; Spaczyński, R.; Pelesz, M. and Pawelczyk, L. (2003): "**Incidence of elevated LH/FSH ratio in polycystic ovary syndrome women with normo- and hyperinsulinemia. **"Division of Infertility and Reproductive Endocrinology. Vol. 48. Pp 131 – 134.**
4. **Barfield ,E; Liu Y; Kessler, M; Pawelczak, M; David ,R and Shah, B. (2009):** "The prevalence of abnormal liver enzymes and metabolic syndrome in obese adolescent females with polycystic ovary syndrome." J PediatrAdolescGynecol .22: 318-322.
5. **Bucolo, G. and David, H. (1973):** "Quantitative determination of serum triglycerides by the use of enzymes." ClinChem; 19(5):476-82
6. **Burtis A et al. (1999):"**Tietz Textbook of Clinical Chemistry, 3rd ed. AACC.

7. **Carmina, E.; Koyama, T.; Chang L.; Stanczyk, F. and Lobo, R. (1992):**"Does ethnicity influence the prevalence of adrenal hyperandrogenism and insulin resistance in polycystic ovary syndrome? Am J ObstetGynecol 1992; 167:1807–1812?
8. **Chang, W.; Knochenhauer, E.; Bartolucci, A. and Azziz, R. (2005):'** Phenotypic spectrum of polycystic ovary syndrome: clinical and biochemical characterization of the three major clinical subgroups". FertilSteril. Jun; 83(6):1717-23.
9. **Cristian, I.; Nicolae, C .and Dan M. (2012):**"Lipid Parameters in Patients with Polycystic Ovary Syndrome. "Applied Medical Informatics Vol. 31(4), pp: 27-32.
10. **Das, M.; Gillott ,D.; Saridogan ,E. and Djahanbakhch, O. (2008):"**Anti-Mullerian
11. **Echiburú, B.; Pérez-B., F.; Maliqueo, M.; Ladrón, d.; Gálvez, C. and Crisosto, N.(2012):"** CAG repeat polymorphism of androgen receptor gene and X-chromosome inactivation in daughters of women with polycystic ovary syndrome (PCOS): relationship with endocrine and metabolic parameters. GynecolEndocrinol 2012;28:516-20.
12. **Ekins, R. (1990)**: Hirsutism: free and bound testosterone Ann.Clin. Biochem 27:91,
13. **Evangeline, V. (2014):** "Nonalcoholic fatty liver disease and polycystic ovary syndrome" World J Gastroenterol. 20(26): 8351-8363.
14. **Evangeline,V. (2014):** "Nonalcoholic fatty liver disease and polycystic ovary syndrome" **World J Gastroenterol. 20(26): 8351-8363.**
15. **Gangale, M.; Miele, L; Lanzone, A; Sagnella, F; Martinez, D; Tropea, A; Moro, F; Morciano, A; Ciardulli, A; Palla, C; Pompili, M; Cefalo, C; Grieco, A and Apa, R. (2011):"**Long-term metformin treatment is able to reduce the prevalence of metabolic syndrome and its hepatic involvement in young hyperinsulinaemic overweight patients with polycystic ovarian syndrome." ClinEndocrinol (Oxf); 75: 520-527
16. **Gerbitz, V.(19980).** J. Clin. Chem. Biochem. 18, 313-326
17. Granulosa cell production of anti-Mullerian hormone is increased inpolycystic ovaries. J ClinEndocrinolMetab. ; 92:240
18. **Haakova L.; Cibula D.; Rezabek K.; Hill M.; Fanta M. and Zivny J. (2003):'** Pregnancy outcome in women with PCO s and in controls matched by age and weight." Hum Reprod. 18:1438–1441.
19. **Hart, R.; Doherty, D.; Norman, R.; Franks, S.; Dickinson, J.; Hickey, M. and Sloboda, D.** Serum antimullerian hormone (AMH) levels are elevated in adolescent girls with polycystic ovaries and the polycystic ovarian syndrome (PCOS). FertilSteril. 2010; 94(3):1118-1121.
20. hormone is increased in follicular fluid from unstimulated ovaries inwomen with polycystic ovary syndrome. Hum Reprod23:2122e 6.
21. **Huerta, R.; Dewailly, D.; Decanter, C.;,Knochenhauer, E.;, Boots, L. and Azziz, R. (1999):**"11-Beta-hydroxyandrostenedione and delta5androstenediol as markers of adrenal androgen production in patients with 21-hydroxylase-deficient nonclassic adrenal hyperplasia". FertilSteril 72:996–1000.
22. **Jonson A.M.; Rohlfs, E.M. and Silverman, L.M. (1999)**.proteins, .In.Burtis, C.A.; Ashwood, E.R. editors. Tietz textbook of clinical chemistry 3rd ed. Philadelphia W.B. Saunders company pp: 477-540.
23. **Joselyn R.; Mervin C.; Luis O.; Milagros R.; Jessenia M.; José M.; MaríaC.andValmore B. (2014):** "Polycystic Ovary Syndrome, Insulin Resistance, and Obesity: Navigating the Pathophysiologic Labyrinth." International Journal of Reproductive Medicine Volume pp: 1-17.
24. **Khayyat, K.; Nahid, A.; Aghdas, S.; and Rasool, R. (2012):**"Body mass index (BMI) related insulin resistance in polycystic ovarian syndrome among patients referred to gynecology clinic of Imam Reza Hospital, Tehran, Iran."Journal of Clinical Medicine and Research Vol. 4(7), pp. 84-88.
25. **Labrie ,F.; Luu-T. V.; Belanger, A.;Lin, SX, Simard, J, Pelletier, G and Labrie, C. (2005):** Is dehydroepiandrosterone a hormone? J Endocrinol. ;187(2):169-96.
26. **Legro, R.; Barnhart, H. and Schlaff, W. (2007):"**Clomiphene, metformin, or both for infertility in the polycystic ovary syndrome.NEngl J Med. 356:551–566.
27. **Lenton, E.; Neal, L. andSulaiman R..(1982):** "Plasma Concentrations of Human Gonadotropin from the time of Implantation until the Second Week of Pregnancy," Fertility and Sterility, (37) 773-78
28. **Mehmet, D.; Ruya, D.; Ozcan, B.; Ummuhani, O.; Eren, A.; Nesat, C.; Nilgun, T.; Mert, K. and Burcu, K.(2015):** "Serum Copeptin, Pentraxin 3, Anti-Mullerian Hormone Levels With Echocardiography and Carotid Artery IntimaMedia Thickness in Adolescents With Polycystic Ovary Syndrome**" J Clin Med Res.(12):989-994**
29. **Meiattini, F.; Prencipe, L.; Bardelli, F.; Giannini, G. and Tarli, P (1978):** "The 4-hydroxybenzoate/4-aminophenazone chromogenic system used in the enzymic determination of serum cholesterol.".Clin Chem. 24(12):2161-5.

30. **Mei-J., C.; Wei-S, Y.; Jehn-H, Y.; Chuhsing , K.; Yu-S., Y. and Hong-Nerng, H. (2006):**"Low sex hormone-binding globulin is associated with low high-density lipoprotein cholesterol and metabolic syndrome in women with PCOS." Human Reproduction Vol.21, No.9 pp. 2266–2271.
31. **Mikola M.; Hiilesmaa V.; Halttunen M.; Suhonen L. and Tiitinen A.(2001):**" Obstetric outcome in women with polycystic ovarian syndrome." Hum Reprod. 16:226–229.
32. **Moran, C.; Knochenhauer, E.; Boots, L. and Azziz R.(1999):** Adrenal androgen excess in hyperandrogenism: relation to age and body mass. FertilSteril 1999; 71:671–674.
33. **Muhammad A. and Nabila R. (2015):**"Endocrine Correlates of Polycystic Ovary Syndrome in Pakistani Women".**Journal of the College of Physicians and Surgeons Pakistan 25 (1): 22-26.**
34. **Muhammad A. and Nabila R. (2015):**"Endocrine Correlates of Polycystic Ovary Syndrome in Pakistani Women". **Journal of the College of Physicians and Surgeons Pakistan 2015, Vol. 25 (1): 22-26.**
35. **Nida, Z. (2014):.**Role of Anti-Mullerian Hormone (AMH) in Polycystic Ovary Syndrome (PCOS)? **Zahid, ReprodSyst Sex Disord 3:4**
36. **Pellatt, L.; Hanna, L.;Brincat, M.; Galea, R.; Brain, H. and Whitehead, S. (2007)**
37. **Praveen, G.; Suresh, G.; Tumu, V.; Himabindu, B.; Mamata, D.; Sisinthy, S.; Manjula, B. (2016):** Analysis of Connexin37 gene C1019T polymorphism and PCOS susceptibility in South Indian population: case–control study Original Research Article. European Journal of Obstetrics & Gynecology and Reproductive Biology, (196) P. 17-20
38. **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. FertilSteril 81: 19-25.
39. **Sanchez L.; Moran, C, Reyna, R; Ochoa, T.; Boots, L.; and Azziz, R.; (2002):**"Adrenal progestogen and androgen production in 21hydroxylase-deficient nonclassic adrenal hyperplasia is partially independent of adrenocorticotropic hormone stimulation. Fertil Steril.77:750–753.
40. **Sayera, B.; Zakir, H.; Fashiur, R. and Laila, A. (2012):** "Polycystic ovarian syndrome in women with acne**.** Journal of Pakistan Association of Dermatologists 22:24-29.
41. **Seddigheh, E.; Maryam, G.; Reza, G. and Mouloud, A. (2015):**"Body Mass Index and Gonadotropin Hormones (LH & FSH) Associate With Clinical Symptoms among Women with Polycystic Ovary Syndrome".Global Journal of Health Science; Vol. 7, No. 2; 2015 ISSN 1916-9736 E-ISSN 1916-9744
42. **Seddigheh, E.; Maryam, G.; Reza, G. and Mouloud, A. (2015):**"Body Mass Index and Gonadotropin Hormones (LH & FSH) Associate With Clinical Symptoms among Women with Polycystic Ovary Syndrome".Global Journal of Health Science; Vol. 7, No. 2; 2015 ISSN 1916-9736 E-ISSN 1916-9744
43. **Seifer, D.; Golub, E.; Lambert-M.,G.; Benning ,L.; Anastos, K. and Watts, D.(2009):"**Variations in serum Mullerian inhibiting substancebetween white, black, and Hispanic women." FertilSteril 92:1674e8.
44. **Setji T.; Holland N.; Sanders L.; Pereira K.; Diehl A.; Brown A. (2006):** "Nonalcoholic steatohepatitis and nonalcoholic Fatty liver disease in young women with polycystic ovary syndrome". J ClinEndocrinolJ ClinEndocrinolMetab. 91(5):1741-7.
45. **Sidhwani, S.; Scoccia, B.; Sunghay, S.; Stephens-A. C.; Mazzone ,T.and Sam, S.(2011):**" PCOS is Associated with Atherogenic Changes in Lipoprotein Particle Number and Size Independent of Body Weight. ClinEndocrinol (Oxf) ;75:76-82.
46. **Sunita, J.; Balasaheb, B.; Jaiprakash, B.; Milind, H.; Varsha, M.; Ravi, G. and N.(1999):**'Hormonal Profile of Polycystic Ovary Syndrome (PCOS) In Indian Women." Research Journal of Pharmaceutical, Biological and Chemical Sciences Volume 3 Issue 4 Page No. 1159.
47. **Vassilatou, E.; Lafoyianni, S.; Vryonidou, A, Ioannidis, D.; Kosma, L.; Katsoulis, K.; Papavassiliou, E. and Tzavara, I. (2010):** Increased androgen bioavailability is associated with non-alcoholic fatty liver disease in women with polycystic ovary syndrome**. Hum Reprod 25: 212-220.**
48. **Wieland, U.D. and Sedidel, J. (1982):** A sample specific method for precipitation of low-density lipoproteins. Lipi. Res., 24:904-909.
49. **Young D.S. (2001):** effects of disease on clinical laboratory test, 4th edition AACC press, Washington D.C.pp. 84-88.