

HORMONAL AND CHEMICAL EVALUATION OF PCOS IN AL-NAJAF, IRAQ

Prof. Hassan Ali Al-Mansoori¹

Article Info

Keywords: Polycystic Ovary Syndrome (PCOS), Hyperandrogenism, Dyslipidemia, Ovulatory Dysfunction, Early Diagnosis

Abstract

Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine and metabolic disorder affecting up to 17.8% of women. It is distinguished by hyperandrogenism, irregular menstrual cycles, and the presence of polycystic ovaries. Hyperandrogenism is a key defining characteristic of PCOS, with around 80% of diagnosed cases meeting the National Institute of Health (NIH) consensus criteria for elevated androgen levels. Hyperandrogenemia is the underlying cause of the distressing signs and symptoms associated with PCOS, including hirsutism, male-pattern baldness, and irregular or absent ovulation. The precise etiology of PCOS remains elusive, but it is closely linked to increased androgen secretion, suggesting aberrant folliculogenesis and steroidogenesis as primary contributors to the condition.

Early diagnosis of PCOS during adolescence is of paramount importance, given its significant association with infertility, obesity, dyslipidemia, diabetes mellitus, cardiovascular disease, and endometrial hyperplasia in later life. Obese individuals with PCOS often exhibit distinctive lipid profiles, characterized by elevated plasma triglycerides and decreased lipoprotein cholesterol concentrations, adding to the complexity of the syndrome. Moreover, PCOS patients may face dyslipidemia due to factors such as obesity, diabetes mellitus, cigarette smoking, and genetic predisposition.

Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrine and metabolic disorder in women[1] with Prevalence of up to 17.8% and is characterized by hyperandrogenism irregular cycles and Polycystic ovaries[2,3]. High Levels of androgen and the criteria's of hyperandrogenism are main features of PCOS. About 80% of polycystic ovarian women who diagnosed by National Institute of Health (NIH) consensus criteria have elevated androgen. Levels [4]. Hyperandrogenemia is directly responsible for the sign and symptom of PCOS which include Hirsutism, acine male pattern baldness and oligo ovulation or Lack of ovulation[5]. The etiology of PCOS is unknown. It is associated with the increased secretion of androgens as constant Protests. It seem that abnormal

¹ University of Kufa, Faculty of Science, Department of Laboratory Investigation

folliculogenesis and steroidogenesis. are the main causes of disease[6]. Despite this difficulties, early Diagnosis of PCOS in adolescence has undeniable importance because this syndrome is significant risk factor for infertility, obesity, dyslipidemia, diabetes mellitus, cardiovascular disease and endometrialhyperplasialater in Life[7]. In obese patients with prof, the Lipoprotein profile is characterized by elevated plasma triglycerides and decrease Lipoprotein Cholesterol concentrations[8]. In addition to dyslipidemia in PCOS, there are several causes such as obesity diabetes mellitus, cigarette smoking and genetic factors[9].

Den to the large increase in women with PCOS and to study the relationship between hormonal and chemical indicators of this syndrome in Iraqi women. This study was conducted which aims to study the hormonal changes accompanying the Syndrome which include FSH LH, prolactin, testosterone, Progesterone and estrogen. In addition this study conducted in order to investigated the effect of PCOS on level of Lipid profile.

Material and Methods Subject of the study

This study included two groups of women. The first group was the PCOS group which included 50 women, with mean age of 29.34 ± 3.21 , PCOS group were collected from Al-Sadr Teaching Hospital in AL- Najaf Iraq within the Period from October 2022 to January 2023 and the Second group was the control group, which included 25 healthy women with mean age of 30.14 ± 3.23 , with regular menstrual cycles.

The body mass index (BMI) determined by multiplying the weight (kg) by the squared height (m²).

The information was collected from the Patients based on special form. that was prepared to Show Some information related to the samples as the effect to these factors and their relationship to polycystic ovary syndrome was studied, age, BMI, Hirsutism, Family history of the patients, number of abortions menstrual period and number of children.

Hormonal assay.

Blood Samples were drawn after an overnight. fast of 12 hours during the early follicular Phase (Cycle day 2 or 3) for evaluation of FSH, LH Prolactin, estrogen and Progesterone in serum, using the commercial kits (BioMerieux Kits). The enzyme linked. fluorescent assay (ELFA) was Performed, using the hormone analyzer (minivids France).

Lipid Profile

The Lipid profile included analysis of measured total cholesterol (TC) triglyceride (TG) and high density lipoprotein (HDL), using Commercial Kits (Biolabo kits). Low density lipoprotein (LDL) and very Low density Lipoprotein (VLDL) were determined in directed, using the Friedewald formula.

Statistical Analysis

It was done by using of SPSS (statistical Package for Social Science) version (21) in which we use frequency with percentage and mean with Standard deviation as description Statistics, for analysis weuse independent Sample t-test. The differences between values were considered Statistically significant at ($P < 0.05$) and ($P < 0.01$).

Results

Basic characteristic of the study group. This study included 50 patients with PCOS obtained from Al-Sadr Hospital in AL-NaJaf. Iraq at the period. between October 2022 to January 2023. These were compared with 25 healthy controls. The percentage of clinical features in women with PCOS are illustrated in. Table (1). According to this table, the percentage of women with PCOS who have hirsutism was (78%) compared to (22%) who did not have hirsutisme. The Family history of the disease (42%) compared to (58%) had no family history of the syndrome, also (62%) of PCOS women suffer from irregular menstrual cycle compared to (38%) who I had a regular menstrual cycle. The same table also shows that women with pros had abortion (34%) compared to (66%)

who did not abortions. A high value of infertility was noted among women with PCOS (68%) compared to (32%) fertile women.

Table (1): Percentage of some clinical features in women with PCOS.

Parameters	Number of Patients	Percentage
Hirsutism (yes)	39	78 %
(No)	11	22%
Family History (yes)	21	42%
(NO)	29	58%
Menstrual periods (irregular)	31	62%
(regular)	19	38%
Abortions (yes)	17	34%
(NO)	33	66%
Infertility (yes)	34	68%
(NO)	16	32%

Table (2): Comparison of the age, BMI and hormone profile between PCOS women and Control group.

	PCOS group Mean $\pm$ SD NO= 50	Control group Mean $\pm$ SD NO = 25	P-value
Age (years)	29.34 $\pm$ 3.21	30.14 $\pm$ 3.23	0.41
BMI (kg/m ²)	31.25 $\pm$ 1.21	24.11 $\pm$ 0.54	0.003
LH (mIU/ml)	13.6 $\pm$ 0.51	4.32 $\pm$ 0.29	0.006
FSH (mIU/mL)	7.71 $\pm$ 0.89	5.12 $\pm$ 0.11	0.01
Prolactin (ng/mL)	14.51 $\pm$ 1.2	6.61 $\pm$ 0.71	0.007
Testosterone (ng/ml)	1.02 $\pm$ 0.10	0.24 $\pm$ 0.02	0.002
Estrogen (E2) (Pg/mL)	51.87 $\pm$ 1.71	92.41 $\pm$ 2.13	0.0016
Progesterone (ng/mL)	6.81 $\pm$ 0.63	13.71 $\pm$ 1.14	0.0012

* Significant difference at show $p < 0.05$ SD Standard deviations.

The results of Table (2) indicate that, There is no significant differences ($p > 0.05$) age between POCS women and control group, also the results show that there is a significant increase ($p < 0.05$) in body mass index (BMI) (31.25 $\pm$ 1.21) kg/m² compared, to control group (24.11 $\pm$ 0.54).

The results from hormonal analysis revealed that the LH and FSH have a significant ($P < 0.05$) Levels ($13.6 \pm 0.51 \mu\text{IU/mL}$; $7.72 \pm 0.89 \mu\text{IU/mL}$ respectively than the control group $4.32 \pm 0.29 \mu\text{IU/mL}$; $5.12 \mu\text{IU/mL}$ respectively in PCOS women. Other parameters such as prolactin and testosterone showed significant increase ($P < 0.05$) Level $14.51 \pm 1.2 \text{ ng/mL}$; $1.02 \pm 0.10 \text{ ng/mL}$ respectively than control group $6.61 \pm 0.71 \text{ ng/mL}$; $0.24 \pm 0.02 \text{ ng/mL}$.

Current results also showed the estrogen and progesterone Levels were significant ($P < 0.05$) in PCOS women ($51.87 \pm 1.71 \text{ Pg/mL}$; $6.81 \pm 0.63 \text{ ng/mL}$ respectively than control group $92.42 \pm 2.13 \text{ Pg/mL}$; $13.71 \pm 1.14 \text{ ng/mL}$).

Table (3) showed that the Lipid profile in PCOS women was measured and compared with healthy control group. This study showed highly significant elevation ($P < 0.01$) in cholesterol, triglycerides, VLDL and LDL in PCOS women ($179.2 \pm 32.3 \text{ mg/dL}$, $154.2 \pm 85.5 \text{ mg/dL}$, $34.2 \pm 17.1 \text{ mg/dL}$ and $115.2 \pm 33.5 \text{ mg/dL}$ respectively, than control group ($152 \pm 31 \text{ mg/dL}$, $112.6 \pm 53.1 \text{ mg/dL}$, $20.9 \pm 9.8 \text{ mg/dL}$ and $78.6 \pm 27.9 \text{ mg/dL}$) while there was a significant decrease ($P < 0.01$) in the level of HDL $40.6 \pm 8.1 \text{ mg/dL}$ in PCOS women compared to ($70.3 \pm 25.7 \text{ mg/dL}$) in control group.

Table (3): Comparison of the Lipid Profile between PCOS women and control group.

Lipid Profile	PCOS group Mean $\pm$ SD No= 50	control group Mean $\pm$ SD No=25	P-value**
Total cholesterol (mg/dl)	179.2 ± 32.3	152.7 ± 31.4	0.001
Triglyceride (TG) (mg/dL)	154.2 ± 85.5	112.6 ± 53.1	0.001
VLDL (mg/dl)	34.2 ± 17.1	20.9 ± 9.8	0.006
HDL (mg/dl)	40.6 ± 8.1	70.31 ± 25.7	0.002
LDL (mg/dl)	115.2 ± 33.5	78.6 ± 27.9	0.007

** significant difference at ($p < 0.01$) SD standard deviations.

Discussion

Current study showed several changes found in clinical features of PCOS women like Hirsutism, Family history recurrent abortion, menstrual Period and infertility (Table 1). Hirsutism occurs as a result of the high concentration of testosterone secreted by the ovaries as a result of the high secretion of LH from the Pituitary gland compared with the FSH, which causes the follicles in the ovaries to produce more testosterone than estrogen (10). The increase in hirsutism in women with PCOS may be due to an increase in androgens secreted by the adrenal gland (11) our results consistent with (12) that among the 93 women with PCOS 40% of them were sisters. Therefore, the prevalence of PCOS among relatives is a positive risk factor, giving information about the progression of PCOS ALSO, hormonal disorders play the main role in the disruption of the menstrual cycle, which leads to stopping ovulation and irregular menstruation (amenorrhea) or absence of menstruation (amenorrhea) and this leads to infertility (13).

It's note that increase in miscarriage cases is associated with an increase in the Level of LH and testosterone and increase in oxidative stress (14) then, obesity and high BMI play risk factor for recurrent abortion (15). A high rate of infertility was noted among women with PCOS, as the increase in the Percentage of infertility may be due to several reasons, including hypothyroidism and an increase in the concentration of TSH, which Leads to an increase in testosterone which affects estrogen (16).

The results in Table (2) indicate that there are no significant difference between the mean ages of women with PCOS and control. If the ages are.. Similar between the two groups and that all ages are within the reproductive age (12-45), then they are at risk of developing polycystic ovary syndrome(17) In addition, both groups PCOS women and control had higher than normal mean, BMI ($<25 \text{ kg/m}^2$) which reflects the fact that obesity plays a major role in the functional and reproductive changes that are Linked with each other. and is considered one of the most important characteristics of PCOS(18).

It was observed through the results of Table (2) an increase in the average concentration of gonadal hormones namely LH and FSH, in women with PCOS at a significant Level ($P<0.05$) compared to the control group, An increase in the rate of refse of hormones releasing gonads feeders Leads to stimulation of the reproduction of the B-subunit of Luteinizing hormone more than FSH (19). Malini and Georges (20) reported that there was difference in the range of LH and FSH Production as well as a higher LH /FSH ratio as the most clinical manifestation of women diagnosed with PCOS.

It is evident from Table (2) a high increase in the Level of the prolactin hormone in the serum of women with PCOS at ($P <0.05$) and this is consistent with what was indicated by Ehrmann (21) who confirmed that the Prolactin hormone suppresses the activity of the aromatase of the granulosa cells of the ovaries and thus supports the theory of the role of prolactin in suppressing Follicular maturation and Lack of ovulation and then Lack of fertilization. It is possible that the rise in proportion of Psychological disorder and nervousness in women with this syndrome or the injury to what is called Anxiety (22). The concentration of testosterone hormone increase significant ($P <0.05$).In women with PCOS and this is consistent. with what was mentioned by Frhan (23) as the concentration of testosterone in the sample of women with PCOS was higher than control, as the increase in LH results from polycystic ovaries, which causes high testosterone and disturbance of other hormones.

Estrogen and progesterone levels are significantly lower in women with PCOS when compared with the control group. This results agreed with chang and Katise)(24) who showed the estrogen hormone Level in PCOS Patients may be Low to normal. other studies also indicated a decrease in the concentration of progesterone in PCOS women, which occurs due to the conversion of progesterone into an androgen before ovulation and that the lack ovulation. prevents the formation of the corpus Luteum, and thus reduced the production! of Progesterone (25).

The Level of the Lipid profile (total cholesterol, triglyceride, very Low density Lipoprotein and Low density Lipoprotein) were presented in Table (3) showed significantly higher in PCOS Patients at ($P<0.01$) than of control group. A significant decline in high density Lipoprotein (HDL) was seen in ($p<0.01$). The reason for dyslipidemia in PCOS may be attributed to hyperinsulinaemia and hyperandrogenemia. This causes adiposities to experience to elevated catecholamine induced Lipolysis and deliver free fatty acids into be the blood elevated free fatty acids in the Liver induce secretion of VLDL which ultimately leads to hypertriglyceridaemia Leads to Low Pathway, hypertriglyceridaemia Leads to Low HDL cholesterol and increased LDL cholesterol levels. It is also possible that hyperandrogenism may also affect Lipid metabolism of HDL particles induction of hepatic Lipase activity which engages in the catabolism of HDL Particles (20) The results of other study by Shoaibet *al*(27) were consistent with the results of the current study, which showed that women with PCOS had a significant increase in Lipid concentration.

Conclusion

PCOS cause Several changes found in clinical features bead to like hirsutism, recurrent abortion menstrual Period, Family history and infertility which play a major role in the occurrence of the syndrome, while excess BMI adversely affected in both PCOS and control, this adverse effect is more prominet in women with PCOS. We found a hormonal imbalance in women caused by PCOS increased Levels of LH, FSH, prolactin, testosterone

and decrease Levels of estrogen and progesteron. The women with PCOS had atherogenic Lipoprotein profile characterized by increased cholesterol, triglycerides, VLDL, LDL especially in obese women which may be a risk factor for developing Cardiovascular complication Later on.

References

- Maliqueo, M., et al (2009). Adrenal function during childhood and puberty in daughters of women with polycystic ovary syndrome. *The Journal of Clinical Endocrinology & Metabolism*, 94(9), 3282-8.
- Azziz, R., et al (2009). The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: the complete task force report. *Fertility and sterility*, 91(2), 456-488.
- March, W. A., et al (2010). The prevalence of polycystic ovary syndrome in a community sample assessed under contrasting diagnostic criteria. *Human reproduction*, 25(2), 544-551.
- Roe, A. H., &Dokras, A. (2011). The diagnosis of polycystic ovary syndrome in adolescents. *Reviews in obstetrics and gynecology*, 4(2), 45.
- Lizneva, D., Gavrilova-Jordan, L., Walker, W., &Azziz, R. (2016). Androgen excess: Investigations and management. *Best practice & research Clinical obstetrics &gynaecology*, 37, 98-118.
- Radosh, L. (2009). Drug treatments for polycystic ovary syndrome. *American family physician*, 79(8), 671-676.
- Moran, L. J., Misso, M. L., et al (2010). Impaired glucose tolerance, type 2 diabetes and metabolic syndrome in polycystic ovary syndrome: a systematic review and meta-analysis. *Human reproduction update*, 16(4), 363-347
- Szendroedi, J., Yoshimura, T., Phielix, et al., (2014). Role of diacylglycerol activation of PKC θ in lipid-induced muscle insulin resistance in humans. *Proceedings of the National Academy of Sciences*, 111(26), 9597.2069
- Khomami, M. B., Tehrani, F. R., Hashemi, S., et al (2015). Of PCOS symptoms, hirsutism has the most significant impact on the quality of life of Iranian women. *PLoS One*, 10(4), e0123608.
- Mofid, A., SeyyedAlinaghi, S. A., Zandieh, S., &Yazdani, T. (2008). Hirsutism. *International journal of clinical practice*, 62(3), 433-443.
- Brown, A. J., Tendler, D. A., McMurray, R. G., &Setji, T. L. (2005). Polycystic ovary syndrome and severe nonalcoholic steatohepatitis: beneficial effect of modest weight loss and exercise on liver biopsy findings. *Endocrine Practice*, 11(5), 319-324.
- Azziz, R., &Kashar-Miller, M. D. (2000). Family history as a risk factor for the polycystic ovary syndrome. *Journal of pediatric endocrinology & metabolism: JPEM*, 13, 1303-1306.
- Hart, R., Hickey, M., & Franks, S. (2004). Definitions, prevalence and symptoms of polycystic ovaries and polycystic ovary syndrome. *Best Practice & Research Clinical Obstetrics &Gynaecology*, 18(5), 671-683.
- Agarwal, A., Aponte-Mellado, A., Premkumar, B. J., Shaman, A., & Gupta, S. (2012). The effects of oxidative stress on female reproduction: a review. *Reproductive biology and endocrinology*, 10, 1-31.

- Wang, J. X., Davies, M. J., & Norman, R. J. (2001). Polycystic ovarian syndrome and the risk of spontaneous abortion following assisted reproductive technology treatment. *Human Reproduction*, 16(12), 2606-2609.
- Dahiya, K., Sachdeva, A., Singh, V., Dahiya, P., Singh, R., Dhankhar, R., ... & Malik, I. (2012). Reproductive hormone and thyroid hormone profile in polycystic ovarian syndrome. *ovarian syndrome. Endocrinology*, 3(6):1-11.
- Chang, J., Azziz, R., Legro, R., Dewailley, D., Franks, S., & Tarlatzis, B. C. (2004). Rotterdam ESHRE/ASRMSponsored PCOS Consensus Workshop Group (2004). Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *FertilSteril*, 81(01), 19-25.
- Inzucchi, S. E., Maggs, D. G., Spollett, G. R., Page, S. L., Rife, F. S., Walton, V., & Shulman, G. I. (1998). Efficacy and metabolic effects of metformin and troglitazone in type II diabetes mellitus. *New England Journal of Medicine*, 338(13), 867-873.
- Coffler, M. S., Patel, K., Dahan, M. H., Malcom, P. J., Kawashima, T., Deutsch, R., & Chang, R. J. (2003). Evidence for abnormal granulosa cell responsiveness to follicle-stimulating hormone in women with polycystic ovary syndrome. *The Journal of Clinical Endocrinology & Metabolism*, 88(4), 1742-1747.
- Malini, N. A., & George, K. R. (2018). Evaluation of different ranges of LH: FSH ratios in polycystic ovarian syndrome (PCOS)—Clinical based case control study. *General and comparative endocrinology*, 260, 51-57.
- Ehrmann, D. A. (2005). Polycystic ovary syndrome. *New England Journal of Medicine*, 352(12), 1223-1236.
- Barry, J. A., Kuczmierczyk, A. R., & Hardiman, P. J. (2011). Anxiety and depression in polycystic ovary syndrome: a systematic review and meta-analysis. *Human reproduction*, 26(9), 2442-2451.
- Farhan, BA. (2003). The effect of IVE Therapy in follicular fluid hormones in female with Luteal phase defects, thesis, MCS, Collage. Med. University. Baghdad.
- Conway, G. (2000). The Polycystic ovary Department of Endocrinology. The Middle sex Hospital Mortimer Street Landon Win8aa. January.
- Swetha, R., Ravi, B. V., Nalini, K. S., Abdelazim, I. A., Elsawah, W. F., Pillai, B. P., ... & Alur, V. C. (2015). Serum lipoprotein (a) and lipid profile in polycystic ovarian syndrome. *J. Clin. Sci. Res*, 4, 2-6.
- Shoaib, O. M., Mustafa, S. M., & Nourein, I. H. (2015). Serum lipid profile of polycystic ovary syndrome in Sudanese women. *International Journal of Medical Science and Public Health*, 4(11), 1.
- Bickerton, A. S. T., Clark, N., Meeking, D., Shaw, K. M., Crook, M., Lumb, 2. P., ... & Cummings, M. H. (2005). Cardiovascular risk in women with polycystic ovarian syndrome (PCOS). *Journal of clinical pathology*, 58(2), 151-154.