

# Research Journal of Pharmaceutical, Biological and Chemical Sciences

## The study of changes in Sex Hormones and Metabolic Parameters clinched alongside ladies with Polycystic ovary syndrome Previously, in Muthanna province – Iraq.

Mohammed Qasim Waheeb<sup>1\*</sup>, Muntthur Mohammad Cani<sup>2</sup>, Hana Ali Aziz<sup>3</sup>

Muthanna University –Iraq<sup>1,3</sup> College of Science<sup>1,3</sup> University of Kerbala-Iraq<sup>2</sup> College of Pharmacy<sup>2</sup>

### ABSTRACT

The present study was concluded in the Feminine & Children Hospital in Samawa city during the period between September 2015 to March 2016. The study was carried out on 60 women that on polycystic ovarian syndrome compared with the control group. Specimens were isolated under two groups, the first group suffering from Polycystic Ovary Syndrome (PCOS) their number (40), the second group control their number (20). Those comes about uncovered a noteworthy expand about low density lipoprotein (LDL) ( $p < 0.53$ ), Body Mass Index (BMI) ( $p < 0.016$ ) and Luteinizing Hormone (LH) ( $p < 0.019$ ). No significant difference in high density lipoprotein (HDL) ( $p < 0.16$ ), Follicle Stimulating Hormone (FSH) ( $p < 0.18$ ), Prolactin (PRL) ( $p < 0.66$ ), Testosterone ( $p < 0.58$ ), total cholesterol ( $p < 0.51$ ) and triglycerides ( $p < 0.05$ ) compared with the control group.

**Keywords** : PCOS, LH, BMI, LDL, Hirsutism.

*\*Corresponding author*

## 1-INTRODUCTION

Polycystic ovary syndrome (PCOS) may be a standout amongst the practically normal endocrine maladies and the practically incessant issue. Previously, ladies from claiming regenerative cell agdistis.. Nowadays, PCOS is in the focus of research because of its increasing prevalence. The etiology of this complex and heterogeneous disorder is still uncertain. Natural variables for example, such that physical inactivity, malnutrition, obesity and insulin resistance (IR) have a crucial role in the development of the disorder [3].

The most common features of PCOS, called the “Rotterdam criteria”, are menstrual disorders (amenorrhoea) such as oligo- or anovulatory menstrual cycles polycystic, large ovaries as detected by ultrasound; and clinical or laboratory signs of excess androgen. It is often associated with obesity, acne, hirsutism, cardiovascular disorders (CVD) and obstructive sleep apnea [24].

PCOS are often accompanied by excessive body weight, with 40-50% of these women being obese (BMI >30 kg/m<sup>2</sup>), and increase in weight worsens the clinical and/or biochemical symptoms. Obesity seen in this population is of the android type, which is a consequence of elevated androgens and insulin resistance, and is associated with an increased waist-to-hip ratio (WHR), type 2 diabetes T2D, and CVD [31].

PCOS is characterized by intraovarian androgen excess, which seems to be responsible for not only the cutaneous manifestations of the syndrome, but also both anovulation and the polycystic appearance of the ovaries. Intraovarian androgen excess stimulates the growth of small follicles, but interferes with the selection of a dominant follicle [17].

In 1935, Stein and Leventhal published a case series of seven women with amenorrhea, hirsutism, and bilateral polycystic ovaries, a condition that later came to be known likewise polycystic ovary syndrome (PCOS). Since its original description, the definition has undergone several revisions [16].

Numerous biochemical parameters and ultrasound appearances together for a mix about clinical features bring been accounted in the analysis for PCOS, but until recently no universally accepted criteria have been adopted for clinical and research purposes [15].

### 1.1-The aim of study:

Those expects about this study, including:

- 1- Measure transforms to biochemical markers for. PCOS ladies toward comparing for the normal ladies.
- 2- Measure transforms on form arrangement also metabolic performance by comparing the body mass index (BMI), metabolic rates using established techniques in women with PCOS.
- 3- Our objective was to determine in the serum of women of hormonal variables in normal and PCOS women.
- 4- The study will also investigate the impact of anthropometric indices such as Body Mass Index (BMI) both PCOS and normal women.

## 2 -MATERIALS AND METHODS

### 2-1-1-Biochemical assays

Biochemical analyses were performed by the Departments of Biochemistry at the Feminine & Children Hospital. Venous blood samples were drawn from an antecubital vein after ultrasound examination.

Samples were collected in plastic sterile tubes, coded to ensure anonymity, centrifuged and serum stored at -20°C until assay.

Serum concentrations of total cholesterol, HDL and triglyceride were measured using an Enzymatic Calorimetric test on a Roche 704 analyzer (Roche Diagnostics Corporation, Indianapolis, USA) with intra-batch COV of 2.0-3.0% and inter-batch COV of 1.7—2.4%.

**2-1-2-Hormonal assays**

Serum levels for LH, FSH, testosterone furthermore prolactin were confirmed by utilizing a the scaled down VIDAS® hormonal analyser (BioMerieux® SA, USA).

**2-1-3-Body Mass Index**

Body mass index or Quetelet index, is factual estimation which compares an individual's weight and more tallness. Body mass index (BMI) is determined mathematically from those tallness furthermore weight measures.  $BMI = \text{weight (kg)} / \text{Height (m}^2\text{)}$ .

BMI values correlate significantly with body fat and obesity, and experts use them to help evaluate a person's health risks associated with underweight[30].

BMI can be categorized as follows, according to the WHO [21]:

- Underweight < 18.5 kg/m<sup>2</sup>
- Normal 18.5 – 24.9 kg/m<sup>2</sup>
- Overweight 25.0 – 29.9 kg/m<sup>2</sup>
- Obese class I, 30.0 – 34.9 kg/m<sup>2</sup>
- Obese class II, 35.0 – 39.9 kg/m<sup>2</sup>
- Obese class III > 40 kg/m<sup>2</sup>

**3. RESULTS**

Sixty (60) women infected PCOS & control. PCOS was numbers (n=40) (**27.1± 4.0**), control was numbers(n=20)(**29.2 ± 2.9**) years. The collection of data was done over a period between September 2015 to March 2016. BMI was the anthropometric parameters analyzed from the data. According to showed normal (BMI **21.9 ± 3.1**kg/m<sup>2</sup>), Overweight PCOS (BMI **23.7 ± 4.5**kg/m<sup>2</sup>).

**Tables (1): Physical, endocrine, biochemical characteristics in controls and PCOS women**

Parameters	Normal (n=20)	PCOS (n=40)	P value
Age, (years)	<b>29.2 ± 2.9</b>	<b>27.1± 4.0</b>	<b>0.38</b>
Body Mass Index(BMI), (kg/m <sup>2</sup> )	<b>21.9 ± 3.1</b>	<b>25.7 ± 4.5</b>	<b>0.016*</b>
Luteinsing Hormone(LH), (IU/L)	<b>5.2 ±2.1</b>	<b>8.6 ± 6.1</b>	<b>0.019*</b>
Follicle Stimulating Hormone(FSH), (IU/L)	<b>5.6 ± 1.2</b>	<b>5.4 ± 1.1</b>	<b>0.18</b>
Prolactin (mU/L)	<b>347 ± 233</b>	<b>390 ± 377</b>	<b>0.66</b>
Testosterone, (nmol/L)	<b>1.38 ±0.78</b>	<b>3.22 ± 0.82</b>	<b>0.58</b>
Total Cholesterol (mmol/L)	<b>4.8 ± 1.6</b>	<b>5.0 ± 1.0</b>	<b>0.51</b>
Triglycerides (mmol/L)	<b>0.73 ± 0.24</b>	<b>1.05 ± 0.7</b>	<b>0.05</b>
High-densitylipoprotein(HDL), (mmol/L)	<b>1.2 ±0.2</b>	<b>1.0 ± 0.3</b>	<b>0.16</b>
Low-densitylipoprotein(LDL), (mmol/L)	<b>3.0 ± 1.6</b>	<b>3.6 ± 0.9</b>	<b>0.53*</b>

\*significant

Values are mean ± SD. Analysis of variance was used to test for a significant increase/decrease from control through PCOS women and p values are given.

Parameters distributed and there was minimal difference between the median and mean values; mean values were therefore compared by ANOVA. The mean value for each parameter respectively, were shown the expected upward trend from control through to PCOS women for BMI, LH, FSH, PRL, testosterone and biochemical assays.

**4. DISCUSSION**

PCOS is a condition in which hormonal imbalances interfere with ovulation. The adrenal glands and ovaries produce excessive amounts of male hormone, which leads to an abnormally high production of luteinizing hormone (LH) also an abnormally low processing of follicle-stimulating hormone (FSH). As a result,

the ovary fills with cysts of immature follicles that are unable to generate eggs. Women with this condition may experience irregular periods, enlarged ovaries, excessive facial and body hair, oily skin, acne and obesity[4].

LH excess has been considered to play a fundamental part in the development of ovarian PCOS. LH hyper-secretion independently or along with insulin was demonstrated to contribute to ovarian hyperthecosis and elevated androgen levels[8][22].

Raised LH pulse plentifulness and increased LH pulse frequency can be observed in two third of PCOS patients because of modified GnRh pulsatility, which reasons a three-fold rise for circle LH versus FSH levels[8]. It was demonstrated in the 1990s that the ovaries played a primary role in the advancement of hyperandrogenemia (HA), As opposed hypothalamic-pituitary system in PCOS [2]. These discoveries negate those starting part of LH in the production of excess androgen in PCOS. LH secretion in PCOS patients shows a decreased hypothalamic affect ability to progesterone negative feedback[23], which might be restored toward flutamide (androgen receptor blocker) [9]. The plasma inhibin and androstenedione concentrations are shown to be correlated, and women with PCOS have elevated levels of serum inhibin-B. Inhibin stimulates androgen production, and in response, androgen stimulates inhibin secretion. This explains the low concentration of follicle-stimulating hormone (FSH) compared to that of LH in anovulatory PCOS women[1]. However, central hypothalamic-pituitary disturbances were determined to be secondary to peripheral ovarian factors[2] and prepubertal hyperandrogenism seems to have an initial role in reduced hypothalamic negative feedback with a rapid GnRh pulsatility[7]. The results of this study agreement with the results, that affirming inability the ovary produce ovarian hormones and lead to low levels in blood against an increase in level androgen hormones that secrete from the ovary [27] [6] [19] [26].

The outcomes from claiming of this study show raised levels of PRL concentrations of patients with PCOS and patients with clinically silent pituitary adenomas. Basal gonadotrophin concentrations are low in most patients with hyperprolactinaemia; most studies suggest that PRL inhibits the release of GnRH, resulting in a state of functional hypogonadotropism[29]. Increasing prolactin levels are frequently associated for disturbances of the menstrual cycle. Most commonly, these are seen in patients with a prolactin-producing pituitary adenoma. Thus, in the initial evaluation process of the infertile patient with irregular menses or amenorrhoea, it is always important to measure prolactin concentrations[10].

PCOS is associated with an adverse cardiovascular risk profile. There is an increase in the conventional danger figures for cardiovascular disease (CVD), including dyslipidaemia (diminished plasma high-density lipoprotein cholesterol (HDL), impaired fibrinolysis, expanded low-density lipoprotein cholesterol (LDL), expanded inflammation, endothelial dysfunction and hypertension and an elevated prevalence of subclinical and clinical atherosclerosis[13][14].

The clear association between obesity and abdominal obesity cannot be overlooked, and this is evident in both adulthood and childhood, on menstrual abnormalities and consequent infertility independent of PCOS. Obesity and abdominal weight done youth also adulthood and weight gain after adolescence are predictors of the development of hirsutism also menstrual disturbances in PCOS[20]. Moreover, women with PCOS constitute a significant proportion of the infertile population. Women with PCOS tend to have a BMI outside the acceptable range (19-25 kg/m<sup>2</sup>) and 40-60% of ladies with PCOS would overweight or obese[18][12]. On a later study comparing ladies with PCOS and age-matched controls compared with the ladies PCOS demonstrated a lower proportion of BMI<25 kg/m<sup>2</sup> and higher proportion of BMI > 30 kg/m<sup>2</sup> and 40 kg/m<sup>2</sup> [11].

Obesity in women is per se a condition of sex hormone imbalance. An increment in body weight may be connected with expanded androgen levels done both ladies with PCOS and in normal controls. [28][25]. For addition, androgens assume a paramount part in the regulation of body fat distribution. Androgen excess in women is generally associated with abdominal obesity, which in turn is a critical determinant of obesity-related metabolic complications[5].

**5. REFERENCE**

- [1] Anderson R, Groome N, Baird D. (1998). Inhibin A: what's more inhibin B in ladies with polycystic ovarian syndrome? Throughout medication with FSH will actuate mono-ovulation. *Clin Endocrinol*.48: 577–577.
- [2] Balen A. (2004). The pathophysiology about polycystic ovary syndrome: attempting with get it PCOS. What's more its endocrinology. *Best Pract Res Clin Obstet Gynaecol*. 18(5): 685-706.
- [3] Baranova A, Tran TP, Biredinc A, Younossi ZM. (2011). Precise review: cooperation for polycystic ovary syndrome with metabolic syndrome and nonalcoholic greasy oil malady. *Sustenance Pharmacol Ther*. 33(7): 801-814.
- [4] Bauer J, Cooper-Mahkorn D (2008). Contraceptive brokenness. Previously, ladies with epilepsy: menstrual cycle abnormalities, fertility, and polycystic ovary syndrome. *Int Rev Neurobiol*; 83:135-55.
- [5] Blouin K, Boivin A, Tchernof A. (2008). Androgens and body fat. Appropriation. *J Steroid Biochem Mol Biol*; 108:272-280.
- [6] Chaudhuri, A. and Marti, B.R. (1998). "Effect of gonadotropin what's more prolactin ahead ovarian movement of a totally ovarian species, the tree pie *Dendrocitta Vagabura* Indian" *J.ExpBiol.*; 36(8):790-5.
- [7] Chhabra S, McCartney CR, Yoo RY, Eagleson CA, Chang RJ, Marshall JC. (2005). Progesterone restraint of the hypothalamic gonadotropin-releasing hormone pulse generator: Proof to shifted impacts in hyperandrogenic juvenile young ladies. *J Clin Endocrinol Metab*. 90: 2810–2815.
- [8] Dumesic DA, Abbott DH, Padmanabhan V. (2007). Polycystic ovary syndrome furthermore its developmental inceptions. *Rev Endocr Metab Disord*. 8(2): 127-141.
- [9] Eagleson CA, Gingrich MB, Pastor CL, Arora TK, Burt CM, Evans WS, Marshall JC. (2000). Polycystic ovarian syndrome: proof that flutamide restores affectability of the gonadotropin-releasing hormone pulse generator to restraint toward estradiol and progesterone. *J Clin Endocrinol Metab*. 85: 4047–4052.
- [10] Ferin, M., Jewelewicz, R. and Warren, M. (1993). *The menstrual cycle: physiology, reproductive disorders and infertility*. New York: Oxford University Press.
- [11] Glueck CJ, Dharashivkar S, Wang P, Zhu B, Gartside PS, Tracy T, Sieve L.(2005). Weight furthermore amazing obesity, manifest toward ages 20-24 years, proceeding through 32-41 years in women, ought caution medical practitioners of the dignostic probability about polycystic ovary syndrome as an reversible underlying endocrinopathy. *Eur J Obstet Gynecol Reprod Biol*;122:206-12.
- [12] Goldzieher JW, Axelrod LR. (1963). Clinical and Biochemical features of Polycystic Ovarian Disease. *Fertile Steril*; 14:631-53.
- [13] Guzick, D.S. (2004). Polycystic ovary syndrome. *Obstetrics & Gynecology*, vol. 103, no. 1, pp. 181 -193.
- [14] Hackney, A.C. (1996). The male reproductive system and endurance exercise. *Medicine and Science in Sports and exercise*, 28, 180-189.
- [15] Homburg, R. What may be polycystic ovary syndrome? a proposition to an agreement on the meaning also analysis of polycystic ovary syndrome. *Hum. Reprod.*; 17: 2495-2499.
- [16] Hsu Roe A, Dokras A. (2002). Those finding of Polycystic Ovary Syndrome in Adolescents. *Rev Obstet Gynecol*. 2011; 4:45-51.
- [17] Jonard S, Dewailly D.(2004). The follicular overabundance on polycystic ovaries, due will intra-ovarian hyperandrogenism, might be those primary offender to those follicular capture. *Hum Reprod Update* 2004; 10:107.
- [18] Kiddy DS, Sharp PS, White DM, Scanlon MF, Mason HD, Bray CS, Polson DW, Reed MJ, Franks S. (1990). Contrasts in clinical and endocrine features the middle of hefty furthermore non-obese subjects with polycystic ovary syndrome: an Investigation of 263 sequential cases. *Clin Endocrinol (Oxf)* .;32:213-20.
- [19] Koracs, G.T. and Norman, R. (2007). "Polycystic Ovary Syndrome" 2nd. Published by Cambridge University.
- [20] Laitinen J, Taponen S, Martikainen H, Pouta A, Millwood I, Hartikainen AL, Ruokonen A, Sovio U, McCarthy MI, Franks A, Jarvelin MR.( 2003). Particular figure span starting with conception to adulthood as An predictor from claiming self-reported polycystic ovary syndrome manifestations.. *Int J Obes Relat Metab Disord*; 27:710-5.
- [21] Laquatra, I. (2004). Nutrition for weight management. In Krause's Food, Nutrition & Diet Therapy. Ed. By L. K. Mahan & S. Escott-Stump. Philadelphia: W.B. Saunders Company. Pp. 558 593.

- [22] Lima MH, Souza LC, Caperuto LC, Bevilacqua E, Gasparetti AL, Zanuto R, Saad MJ, Carvalho CR. (2006). Up-regulation of the phosphatidylinositol 3-kinase/protein kinase b pathway in the ovary of rats by incessant medicine for hCG and insulin. *J Endocrinol.* 190(2): 451-459.
- [23] Marshall J, Eagleson C, McCartney C. (1999). Neuroendocrine parts of polycystic ovary syndrome. *Endocrinol Metab Clin North Am.* 28: 295–324.
- [24] Nitsche K, Ehrmann DA. (2010). Obstructive rest apnea furthermore metabolic brokenness to polycystic ovary syndrome. *Best Pract Res Clin Endocrinol Metab.* 24(5): 717-730.
- [25] Pasquali R. Corpulence and androgens: Realities furthermore perspectives. *Fertil Steril* (2006); 85:1319-1340.
- [26] Sahin, Y.; Unluhizarci, K.; et al. (2007)."The impacts metformin with respect to metabolic and cardiovascular danger figures over nonobese ladies with polycystic ovary syndrome" *Clin.Endocrinol. (Oxf).* ; 67(6):904-8.
- [27] Scott, M.G.; Ladson, J.H.; et al(1989)."Hormonal assessment from claiming female fruitlessness and conceptive disorders"*Clint.Chem.:* 35:620-9.
- [28] Taponen S, Martikainen H, Jarvelin M-R, Laitinen J, Pouta A, Hartikainen A-L, Sovio U, McCarthy MI, Franks S, Ruokonen A. (2003).Hormonal Profile for ladies with Self-Reported indications of oligomenorrhea or hirsutism:Northern Finland conception companion. 1966 Study. *J Clin Endocrinol Metab;* 88:141-147.
- [29] Tietz (2006). Reading material about clinical science chemistry and Molecular Diagnostics, 4th Edition, Elsevier Saunders Publishers, pp2021-2027.
- [30] Whitney, E. N. and Rolfes, S. R. (2002). *Understanding Nutrition.* 9th ed. Australia: Wadsworth.
- [31] Yildiz B, Bozdogan G, Yapici Z, Esinler I, Yarali H.(2012). Prevalence, phenotype, and cardiometabolic danger of polycystic ovary syndrome under different symptomatic criteria. *Hum Reprod*,27:3067-3073.