International Journal of Sciences: Basic and Applied Research (IJSBAR)

ISSN 2307-4531
(Print & Online)

http://gssrr.org/index.php?journal=JournalOfBasicAndApplied

Hormonal Changes in Women Patients with Polycystic Ovary Syndrome (PCOS)

Nagham Hasan Ali*

College of Dentistry / Tikrit University

Abstract

In Iraqi women theirs problem related to polycystic ovary syndrome, therefor the present study focus in these women patients and aimed to determine the hormonal changes in the class of these women the hormonal changes included Follicle-stimulating hormone FSH, Luteinizing hormone LH, and Prolactin (PRL) and study the correlation between theme using statistical analysis, to achieve good results from current research 30 specimens of blood were collected from 45 patients with PCOS and 30 healthy between 27 and 44 old year's consider- as control samples attending the private clinic in Baghdad governorate from the period November 2015 to June 2016. All samples were inoculated to study different characteristics of hormones. The results obtained from statistical analysis appeared that there's significant difference between the studied hormones $P \geq 0.05$ also there's high significant difference in the titration of the same hormone in the patients based on the age of women patients' blood was hormonal assay results revealed a gradual decrease in serum FSH levels with a concomitant increase in Serum LH hormone level , when compared with control healthy also there's depression in Prolactin in thyroid cancer patients Compared with control healthy.

***Keywords*:** PCOS; hormonal changes; ELIsA.

---

* Corresponding author.

1. Introduction

Polycystic ovary syndrome,(PCOS) is the most common cause of anovulation, Infertility and hyper and rogenism in women, affecting between 5 and 10% of women of reproductive age worldwide[1]. Chronic anovulation is a common disorder in women of reproductive age. Some women with chronic anovulation also have high androgen levels or polycystic ovary appearance on ultrasound examination, and are diagnosed as having polycystic ovary syndrome (PCOS) by the 2003 Rotterdam criteria [2]. The pathogenesis of chronic anovulation, with or without PCOS, is poorly understood. A high incidence of similar phenotypes in family members of PCOS patients suggests that genetics may play a role [3]. Though the polymorphisms in genes encoding sex hormones and their receptors have been investigated, results are still conflicting [4,5]. Assessing hormone levels serves two major purposes. First of all, it helps to rule out any other problems that might be causing the symptoms. Secondly, together with an ultrasound and personal and family histories, it helps your doctor confirm that you do have PCOS . Most often, the following hormone levels are measured when considering a PCOS diagnosis: Lutenizing hormone (LH), Follicle Stimulating hormone (FSH), Total and Free Testosterone , Dehydroepiandrosterone sulfate (DHEAS) , Prolactin, rostenedione and Progesterone. LH , FSH and prolactin are the hormones that encourage ovulation[6]. Both LH and FSH are secreted by the pituitary gland in the brain. At the beginning of the cycle, LH and FSH levels usually-range between about 5-20 mlU/ml. Most women have about equal amounts-of LH and FSH during the early part of their cycle. However, there is a LH-surge in which the amount of LH increases to about 25-40 mlU/ml 24 hours before ovulation occurs . Once the egg is released by the ovary, the LH levels goes back down [7].

While many women with PCOS still have LH and FSH still within the 5-20 mlU/ml range , their LH level is often two or three-times-that-of-the FSH-level . It is typical for women with PCOS to have an LH level of about 18 mlU/ml and FSH-level-of about 6 mlU/ml (both levels fall within the normal range of 5-20 mlU/ml)[8]. This situation is called an elevated LH to FSH ratio or a ratio of 3:1 . This change in the LH to FSH ratio is enough to disrupt ovulation. While this used to be considered-an important aspect in diagnosing PCOS , it is now considered less useful in diagnosing PCOS , but is still helpful when looking at the overall picture [9].

The current study designed to investigate some hormonal changes of polycystic ovary syndrome women patients based on hormonal abnormality and compared with control.

2. Material and Methods

Thirty (30) specimens of blood were collected from 45 women patients with Polycystic ovary syndrome (PCOS) and 30 healthy between 27 and 44 years old attending the Private clinics hospital from the period from November 2015 to June 2016. All samples were inoculated to study different characteristics of hormons. At the experiment blood samples were drawn and serum was collected by centrifugation. Follicle-stimulating hormone (FSH) concentrations were measured and Lutenizing hormone (LH), and Prolactin in serum by ELIZA kits (Beckman-France), as presented in standard Assay [10] procedures respresented in figure 1 . The results were analyzed using one-way analysis of variance (ANOVA), the level of statistic was set at $P<0.05$(11).

Procedure for FSH, mIU/ml

Format the micro plate wells for each serum reference, control and patient specimen

⇩

- *Add 50µL of the appropriate serum reference or specimen into each well*

⇩

Incubation 15 minutes at room temperatures

⇩

Add50µl stop solution to each

⇩

Swirl the micro plate 20-30 second

⇩

Read the absorbance in each wells at 450 nm

Procedure for LH

Format the micro plate wells for each serum reference, control and patient specimen

⇩

- *Add 25µl of the appropriate serum reference or specimen into each well*

⇩

Add 100µl of working reagent A, LH *enzyme reagent to all wells*

⇩

Swirl the micro plate 20-30 second

⇩

Incubation 60 minutes at room temperatures

⇩

Discard the contents of the micro plate

Swirl the micro plate 20-30 second

⇩

Read the absorbance in each wells at 450 nm

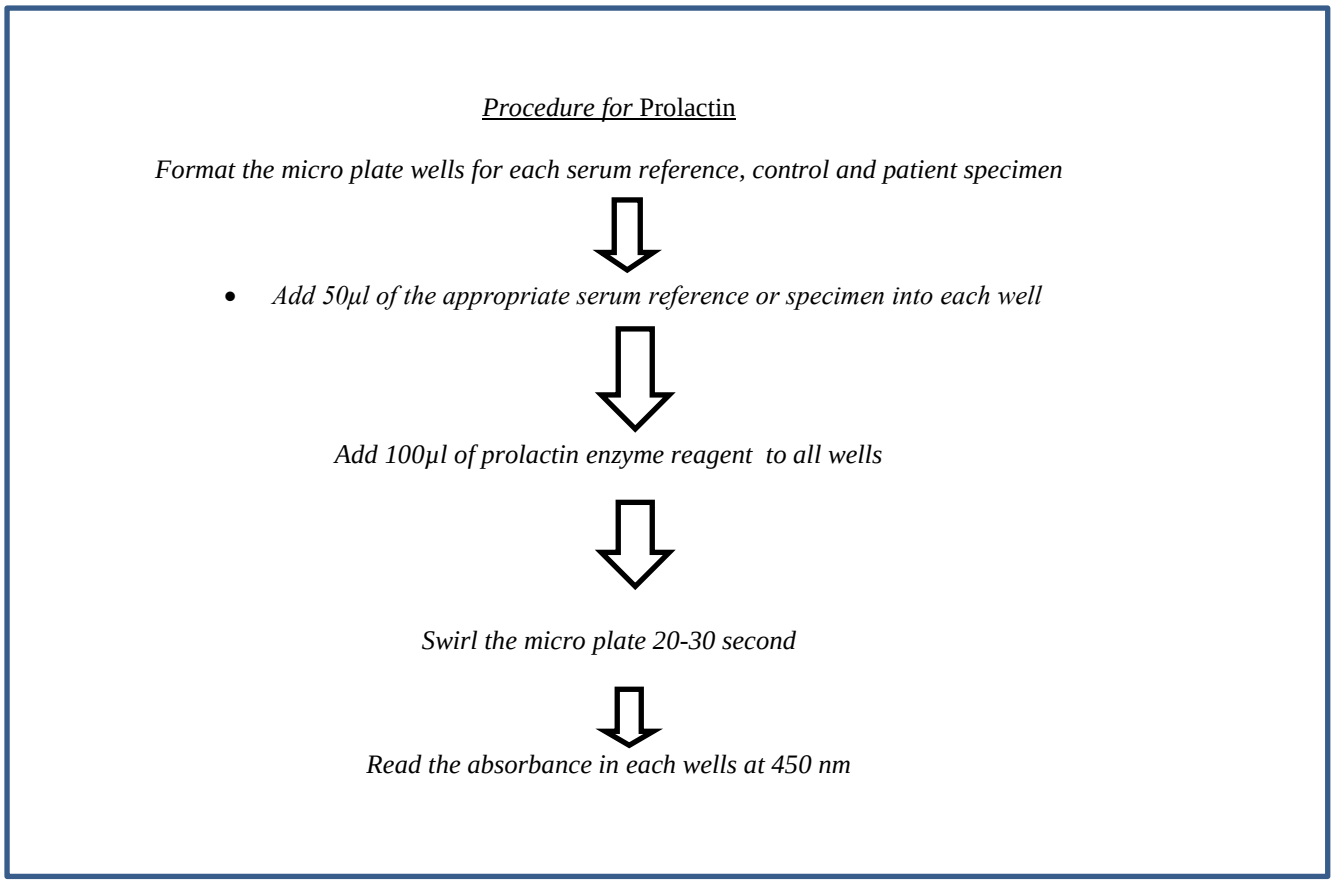

Figure 1: procedure of hormones analysis subjected during the present study

Statistical Analysis:

The values of the investigated parameters were given in terms of mean ± standard error, and differences between means were assessed by analysis of variance (ANOVA) using SAS computer program version 7.5. Differences in results were considered significant at probability value equal or less than 0.05 and 0.001.

3. Results and Discussion

Our findings support to the relationship with hormonal changes. To study the disorder of hormones in the women patients with PCOS The analysis of blood subjected and appeared that theirs significant difference between the healthy group and women with PCOS patients during the period of study as appeared in table (1).

Hormonal changes play important roles in the patients with PCOS Table (1) Represented and summarized the hormonal profile of women with -PCOS subjected in the study compared with healthy in the study groups the mean value concentration of FSH mIU/ml in control 6.52mIU/ml while the average in patients groups reach to 16.0 mIU/ml the LH mIU/ml concentrations in PCOS patients between 6.9-21.4.The results showed a general trend for FSH values to increase in patients group, this increase

reached statistical significance (P<0.05) where the average values of serum Prolactin in control group was 9.2 while the average concentrations in patients was 5.61 its ranged between 1.4 to 12.3 results appeared that prolactin decrease in patients when compared with the control.

Table 1: Average of Follicular stimulating hormone (FSH) , (LH) and (Prolactine) in women patients with PCOS compare with healthy control.

NO.	FSH, mIU/ml	LH mIU/ml	Prolactine mIU/ml
Average	19.0±0.04	8.61±0.05	49.9±0.05
Control			
Average	12.5±0.02	11.2±0.01	61±0.05

Figures 2,3 and 4, summarize the concentrations profile of the three studied hormones FSH, mIU/ml, LH3 mIU/ml and prolactin in women patients with polycystic ovary syndrome (PCOS).

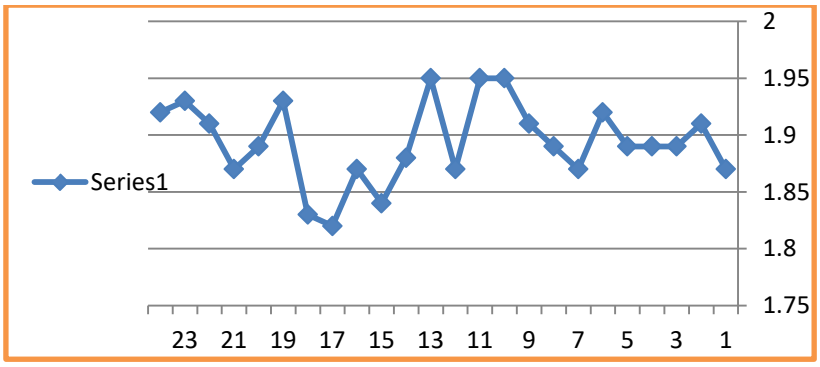

Figure 2: Follicle-stimulating hormone (FSH) in women patients with PCOS compared with healthy.

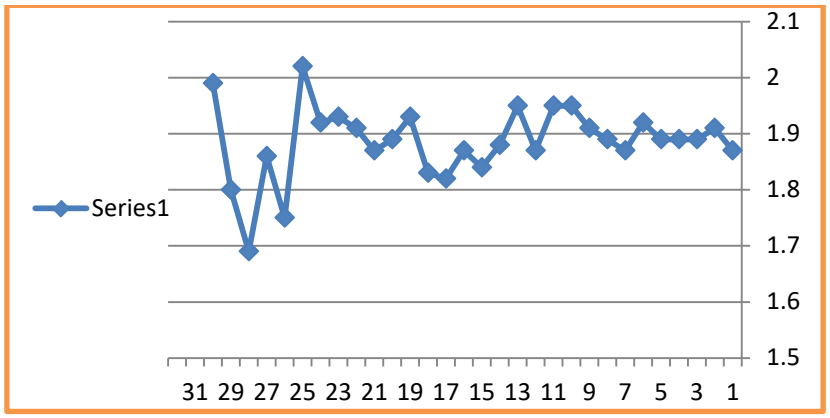

Figure 3: Lutenizing hormone (LH) mIU/ml in patients with PCOS compared with healthy.

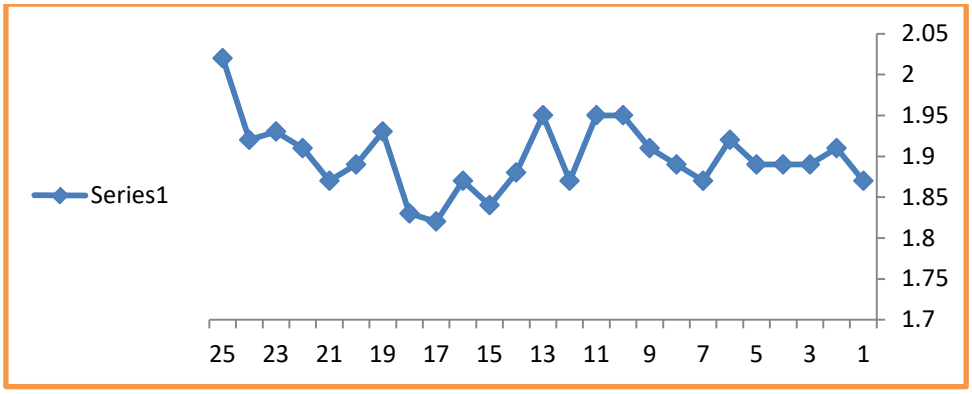

Figure 4: Prolactin hormon mIU/ml in patients with PCOS compared with healthy.

Statistical correlations of Follicle-stimulating hormone FSH, Luteinizing hormone LH, and Prolactin (PRL)

Our results clear the mode of correlations between the Follicle stimulating hormone FSH, Luteinizing hormone LH, and Prolactin (PRL) in the PCOS women patients. Studied statically according to the Pearson correlation as shown at the table (2), there was a significant correlation between studied hormones (p 0.001).

Table 2: The correlations between the three Follicle stimulating hormone FSH Luteinizing hormone LH, and Prolactin (PRL) hormoes

Marker		FSH	LH	PRL
FSH	Pearson Correlation	_	0.348 ***	-
	Sig.(2-tailed)	_	0.005	-
	No.	30	30	30
LH	Pearson Correlation	0.348 ***	_	23.09
	Sig.(2-tailed)	0.005	_	37.9
	No.	30	30	30
PRL	Pearson Correlation	23.4	11	0.348 ***
	Sig.(2-tailed)	45.9	39	0.005
	No.	30	30	30

***P <0.001

**Pearson's correlation between two variables is defined as the covariance of the two variables divided by the product of their standard deviations.

Our present results may be related to Polycystic ovary syndrome (PCOS) represents the most common endocrinopathy of women of reproductive age [12]. Although descriptions of polycystic ovaries date back to the early 1700s, variable degrees of virilization, menstrual abnormalities, and bilaterally enlarged polycystic ovaries form the basis for the definition of PCOS [13]. In addition to the clinical manifestations of infertility and virilization, metabolic consequences of PCOS include obesity [14]. To date, there is no overall consensus on the definition of PCOS or the criteria used for diagnosis [15]. PCOS is a disorder of androgen excess or hyperandrogenism with the following clinical findings: hyperandrogenism (hirsuitism and/or hyperandrogenemia), ovarian dysfunction (oligo-anovulation and/or polycystic ovaries), and the exclusion of other androgen excess related Disorders of hormones [16]. The area of active investigation in PCOS has been the ovary [17]. This analysis has focused primarily on the granulosa and theca interstitial cells of the small Graafian follicles that accumulate in the cortex of PCOS ovaries [18].

4. Conclusion

Our study showed that there was significant difference between PCOSr patients and healthy human at the studied group may be due to molecular changes that it is more precise and less consuming time in study of thyroid cancer and this result may help in control and develop treatment for these cases.

References

[1] Ehrmann DA: Polycystic ovary syndrome. N Engl J Med 2005, 352:1223–12236.

[2] Norman RJ, Masters S, Hague W: Hyperinsulinemia is common in family members of women with polycystic ovary syndrome. Fertil Steril 1996, 66:942–947.

[3] Simoni M, Tempfer CB, Destenaves B, Fauser BC: Functional genetic polymorphisms and female reproductive disorders: Part I: Polycystic ovary syndrome and ovarian response. Hum Reprod Update 2008, 14:459–484.

[4] Gromoll J, Simoni M, Nordhoff V, Behre HM, De Geyter C, Nieschlag E: Functional and clinical consequences of mutations in the FSH receptor. Mol Cell Endocrinol 1996, 125:177–182.

[5] Aittomaki K, Lucena JD, Pakarinen P, Sistonen P, Tapanainen J, Gromoll J, Kaskikari R, Sankila EM, Lehvaslaiho H, Engel AR, Nieschlag E, Huhtaniemi I, De la Chapelle A: Mutation in the follicle-stimulating hormone receptor gene causes hereditary hypergonadotropic ovarian failure. Cell 1995, 82:959–968.

[6] Gromoll J, Simoni M: Genetic complexity of FSH receptor function. Trends Endocrinol Metab 2005, 16:368–373.

[7] Conway GS, Conway E, Walker C, Hoppner W, Gromoll J, Simoni M: Mutation screening and isoform prevalence of the follicle stimulating hormone receptor gene in women with premature ovarian failure, resistant ovary syndrome and polycystic ovary syndrome. Clin Endocrinol (Oxf) 1999, 51:97–99.

[8] Tong Y, Liao WX, Roy AC, Ng SC: Absence of mutations in the coding regions of follicle-stimulating hormone receptor gene in Singapore Chinese women with premature ovarian failure and polycystic ovary syndrome. Horm Metab Res 2001, 33:221–226.

[9] Sudo S, Kudo M, Wada S, Sato O, Hsueh AJ, Fujimoto S: Genetic and functional analyses of polymorphisms in the human FSH receptor gene. Mol Hum Reprod 2002, 8:893–899.

[10] Katz P, Bellet N, less M.CEDIATM total an automated homogenous enzyme-immunoassay for serum total fsh,testosteron. Clin Chem.1991;37:9936.

[11] Steel, RG. and Torries JH. (1980). Principal and Procedures of Statistics. Abiometrical approach 2nd Ed. McGraw - Hill Book Company. New York USA.

[12] Overbeek A, Kuijper EA, Hendriks ML, Blankenstein MA, Ketel IJ, Twisk JW, Hompes PG, Homburg R, Lambalk CB: Clomiphene citrate resistance in relation to follicle-stimulating hormone receptor Ser680Ser-polymorphism in polycystic ovary syndrome. Hum Reprod 2009, 24:2007–2013.

[13] Carmina E, Koyama T, Chang L, Stanczyk FZ, Lobo RA: Does ethnicity influence the prevalence of adrenal hyperandrogenism and insulin resistance in polycystic ovary syndrome? Am J Obstet Gynecol 1992, 167:1807–1812.

[14] Saiki, R. 1989. Chapter 1. In PCR Technology: Principles and Applications for DNA Amplification, H. A. Erlich (Ed.). Stockton Press, New York

[15] Knochenhauer ES, Key TJ, Kahsar-Miller M, Waggoner W, Boots LR, Azziz R: Prevalence of the polycystic ovary syndrome in unselected black and white women of the southeastern United States: a prospective study. J Clin Endocrinol Metab 1998, 83:3078–3082.

[16] Diamanti-Kandarakis E, Kouli CR, Bergiele AT, Filandra FA, Tsianateli TC, Spina GG, Zapanti ED, Bartzis MI: A survey of the polycystic ovary syndrome inthe Greek island of Lesbos: hormonal and metabolic profile. J Clin Endocrinol Metab 1999, 84:4006–4011.

[17] Wijeyaratne CN, Balen AH, Barth JH, Belchetz PE: Clinical manifestations and insulin resistance (IR) in polycystic ovary syndrome (PCOS) among South Asians and Caucasians: is there a difference? Clin Endocrinol (Oxf) 2002, 57:343–345.

[18] Vutyavanich T, Khaniyao V, Wongtra-Ngan S, Sreshthaputra O, Sreshthaputra R, Piromlertamorn W: Clinical, endocrine and ultrasonographic features of polycystic ovary syndrome in Thai women. J Obstet Gynaecol Res 2007, 33:677–680.

[19] Lussiana C, Guani B, Mari C, Restagno G, Massobrio M, Revelli A: Mutations and polymorphisms of the FSH receptor (FSHR) gene: clinical implications in female fecundity and molecular biology of FSHR protein and gene. Obstet Gynecol Surv 2008, 63:785–789.