

# Hormonal Study for Iraqi Women with Polycystic Ovary syndrome

Shahad Abbas Hameed<sup>1\*</sup>, Fatin Fadhel Mohammed Al-Kazazz<sup>2</sup>, Ahmed Mahdi Rheima<sup>3</sup>

<sup>1</sup>Department of Chemistry, College of Science, Mustansiriyah University, Baghdad, Iraq

<sup>2</sup>Department of Chemistry, College of Science, Mustansiriyah University, Baghdad, Iraq

<sup>3</sup>Department of Chemistry, College of Science, Mustansiriyah University, Baghdad, Iraq

Email: [ahmed.rheima@gmail.com](mailto:ahmed.rheima@gmail.com)

\*Corresponding author: Ahmed Mahdi Rheima [ahmed.rheima@gmail.com](mailto:ahmed.rheima@gmail.com)

---

**Received:** 16 January 2023

**Accepted:** 7 April 2023

---

**Citation:** Hameed SA, Al-Kazazz FFM, Rheima AM (2023) Hormonal Study for Iraqi Women with Polycystic Ovary syndrome. *History of Medicine* 9(1): 1913–1917. <https://doi.org/10.17720/2409-5834.v9.1.2023.245>

---

## Abstract

Ovarian polycystic disorder (PCOS) is a common a main hyperandrogenic disorder in which women exhibit hormonal imbalance Ovarian cysts develop when the sex hormones progesterin and estrogen are present and are out of balance. This syndrome causes disorders of the female reproductive system due to hormonal disorders, which tiny ovarian cysts on ultrasonography of the ovaries reveal. Among the elements that can impact the disease's clinical signs include obesity and insulin resistance. Therefore, in the current study, we sought to better understand the associations between certain body composition measures and hormone concentrations (including total testosterone, follicle-stimulating hormone, and luteinizing hormone) in a sample of women with and without PCOS. Patients and Methods: 120 blood specimens were collected from Iraqi women attending Private AL-Batoul Hospital for Obstetrics and gynecology. The samples were divided into 60 patients and 60 healthy control groups. Hormones analysis was performed by using an ELFA (enzyme-linked fluorescent assay) method. Results: The data showed a non-significant alteration in the level of FSH for PCOS patients ( $p > 0.05$ ) in comparison to a healthy control group. According to BMI, a significant difference was between the studied groups, while mean levels of LH and LH/FSH, PRL, and testosterone were increased significantly for both groups of PCOS patients (had child and no child) in comparison to the control group. Conclusion: -PCOS can consider a disorder in the androgenic hormones furthermore the gonadal atropine hormones.

---

## keywords

Polycystic ovary syndrome, PCOS, androgenic hormone, LH/FSH ratio

---

Polycystic ovarian syndrome (PCOS): is a main hyperandrogenic disorder in which women present with unbalanced hormone levels The imbalance of the sex hormones estrogen and progesterone causes the development of ovarian cysts. It is the most common problem in female endocrine systems throughout reproductive age, with currently unknown diagnostic criteria and inadequate sample methods [1,2] Polycystic ovary syndrome affects one in every ten women of reproductive age (PCOS). In women with PCOS, erratic hormones and digestive problems are common. [3,4]. 8-13% of women of reproductive age are thought to be affected by this disorder, which is one of the most prevalent. Insulin resistance, Pregnancy issues

(early pregnancy loss, prenatal hypertension, and preeclampsia/eclampsia), hirsutism, infertility, and irregular menstrual periods) are just a few of the symptoms and indicators that PCOS patients deal with (IR), metabolic syndrome, pre-diabetes, and type 2 diabetes (DM2) Their lifelong medical needs are mostly driven by anxiety and despair. [5,6]

Stein and Leventhal originally identified the condition known as polycystic ovarian syndrome (PCOS) as an amenorrhea syndrome in 1935., or oligomenorrhoea , hirsutism, obesity, and clinical characteristics have been associated with polycystic ovarian syndrome[1]

The Rotterdam criteria are now part of the extended PCOS diagnostic criteria. by the international

consensus European Society for Human Reproduction and Embryology/American Association for Reproductive Medicine (ESHRE/ASRM) workshop group\, which is based on the fact that women who have two of the following three criteria: (1) irregular menstruation (infrequent menstrual cycles) and/or anovulation; (2) clinical and/or biochemical hyperandrogenic; (3) ultrasound-detected polycystic ovaries [7]. The NIH created diagnostic criteria for PCOS in 1990, which include excessive levels of male sex hormones and irregular periods but forbid other conditions that resemble PCOS, such as adult-onset hyperprolactinemia, Androgen-emitting tumors, and inborn adrenal hyperplasia. The Endocrine Society states,) women who experience two of the associated symptoms of PCOS—excessive androgen production, should be assessed, along with anovulation and pearl-sized blisters in the ovaries. —should be evaluated [8,9]. Luteinizing hormone (LH) is characterized as a hormone glycoprotein, co-secreted by the gonadotrophin cells together with the follicle-activating hormone in the adenohypophysis (anterior pituitary) [10] Lutropin, often known as LH or luteinizing hormone, is a glycoprotein hormone made up of two distinct subunits. LH has a molecular weight of around 29 kilograms [11]. The level of LH is important in the ovulation process releasing the egg, [7] LH belongs to a neurological pathway consisting of the pituitary gland, the hypothalamus, and the gonads. In this prescribed pathway, LH release is activated by the gonadotropin-releasing hormone (GnRH). LH performs different functions, as related to women and men. LH in both sexes makes its contributes to primordial germ cell maturation[4]. Follicle-stimulating hormone The basophilic cell's gonadotropes of the anterior pituitary gland produce and synthesize the hormone that stimulates follicles (FSH), which is classified as a glycoprotein [12]. FSH controls pubertal development, human reproductive functions, as well as growth, and development. In terms of reproduction, FSH and LH are comparable. Follicle development, corpus luteum creation, ovulation, and the synchronized release of progesterone and estradiol are all triggered by LH and FSH[12]. Testosterone hormone Anabolic steroid testosterone is (17 Я-hydroxyandrost-4-en-3-one) primarily produced by Leydig cells in the ovary, testes OF meal, and adrenal glands of both sexes. [13], is a cholesterol-based anabolic-androgenic steroid that is produced mainly in men's Leydig cells, and women's adrenal zona fasciculata (via conversion from progesterone) and ovary (25%) as well as the adrenalzona fasciculata (through conversion from progesterone), with the remaining 50% originating from circulating androstenedione. [14]. High ovarian androgen secretion is the major abnormal hormonal feature of polycystic ovary syndrome[6]. Prolactin Hormone (PRL) is a polypeptide that consists of (199) amino acids comprising 3 disulfide intramolecular

bonds, it is important for the survival of huma[16] The versatile polypeptide hormone prolactin (PRL) plays a variety of roles in physiology, including those related to metabolism, growth, and development, immunity, brain and behavior regulation, and reproduction [47]. Pituitary lactotroph cells release PRL. antagonists of the dopamine, estrogen and thyrotropin-releasing hormone (TRH) receptors all work together to promote the generation of PRL. [15].

This study aims to evaluate the gonadal hormones related to androgens hormones in sera of Iraqi women with polycystic ovaries at Wasit Governorate, due to the increasing incidence of this syndrome during the last five years, (in the year 2022 was about 2060 while in 2017 about 364)

## Material and Methods

### Subjects

This research has been achieved at AL-Batoul Hospital for Obstetrics and gynecology from June 2022 to September 2022 . . The study includes 120 Iraqi women divided into two groups 60 PCOS patients and 60 healthy controls. The medical history of all PCOS patients has been taken and has been detected first by ultrasound scan, supported by information recorded if they experienced oligomenorrhea, amenorrhea, or highly irregular menses. The age range for patients is between 16-42 years. The patient is identified if any two of the following occur existed:

- Polycystic ovary syndrome presences on ultrasound scan.
- Signs of clinical and/or biochemical hyperandr ogenism.

### Collection of samples

Blood Specimens have been collected, by taking 6 ml of venous blood from each PCOS patient and healthy Women were recruited from each instance between the second and fifth day of the early follicular phase (for those with a regular cycle). Clot activator tubes were used to collect blood samples, and the serum was separated by centrifugation for 10 minutes at 3000 xg and divided in. Eppendorf tubes are used to prevent repeated freezing and thawing and are kept frozen until analysis.

## Methods

Hormones analysis was performed by using the enzyme-linked fluorescent assay (ELFA) technique (references company components of the (LH, FSH, testosterone, and PRL) Kit materials supplied

- 1) Package for cartridges:
- a) Cartridges 25.

- b) 35 L 25 Capillary tube.  
 c) ID Chip 1.  
 2) Boundary Box:-  
 a) Detectors 25.  
 b) AFIAS-50 Diluent 1

## Results and Discussion

The demographic study for subjects was illustrated in Table (1). There is a non-significant variance

( $P > 0.05$ ) in the mean of age between PCOS with and without children ( $28.3 \pm 6.1$ ,  $26. \pm 6.5$ ,) respectively compared to healthy control groups ( $27.71 \pm 5.9$ ). Mean BMI is a highly significant increase ( $P < 0.001$ ) for patients with children ( $35.4 \pm 15$ ) in comparison with controls ( $24.1 \pm 2.61$ ), as shown in Table 1. The result indicates that the mean was a significant increase ( $p < 0.05$ ) with no child PCOS patients ( $29.45 \pm 6.8$ ) compared with the mean of the control group ( $24.1 \pm 2.61$ )

Table 1:- Distribution of PCOS patients and controls according to age, Weight, Height

Parameters	Control, N= 60 (mean $\pm$ SD)	Patients with child N=30 (mean $\pm$ SD)	Patients with no child N=30 (mean $\pm$ SD)
Age (year)	28.3 $\pm$ 6.1,	26 $\pm$ 6.5	27.71 $\pm$ 5.9
BMI (kg/m <sup>2</sup> )	24.1 $\pm$ 2.61	35.4 $\pm$ 15 <sup>c**</sup>	29,45 $\pm$ 6.8 N*
Weight(kg)	65 $\pm$ 8.1	81.2 $\pm$ 12 <sup>c**</sup>	75. 8 $\pm$ 13 N*
Height (m)	156 $\pm$ 6	156 $\pm$ 18	159.8 $\pm$ 6

\*  $p < 0.05$  Significant; \*\*  $p$  highly Significant

c compare between control and patients with child; N compare between control and patients with no child, M compare between patients with child and patients with no child. BMI: Body Mass Index= weight/ (height)<sup>2</sup>,

the most typical endocrine condition affecting women of reproductive age is PCOS. It has been well established over the past 30 years that insulin

resistance is a significant contributor to the pathophysiology of PCOS's metabolic and reproductive problems. a disorder affects 5% to 10% of women of reproductive age with symptoms (12–45 years old). It is believed to be the most common endocrine issue among women of reproductive age and one of the main reasons for female infertility [21].

Table (2): Comparison between PCOS patients and controls groups according to the selected Hormonal profile

Parameters	Control group N= 60 (mean $\pm$ SD),	PCOS patients with child N=30 (mean $\pm$ SD)	PCOS patients with no child N=30 (mean $\pm$ SD)
LH (IU/ml)	5.4 $\pm$ 2.1	7.8 $\pm$ 3.2 <sup>c**</sup>	5.4 $\pm$ 2.5 <sup>N*</sup>
FSH (IU/ml)	5.1 $\pm$ 1.8	5.3 $\pm$ 3.0	4.9 $\pm$ 2.6
LH/FSH ratio	0.17 $\pm$ 0.19	1.9 $\pm$ 1.04 <sup>c**</sup>	1.18 $\pm$ 0.46 <sup>N**</sup>
PRL (IU/ml)	18.0 $\pm$ 6.3	37.2 $\pm$ 23.1 <sup>c**</sup>	30.0 $\pm$ 28 <sup>N*</sup>
Testosterone (ng/ml)	0.20 $\pm$ 0.04	0.58 $\pm$ 0.069 <sup>M**</sup>	0.51 $\pm$ 0.09 <sup>M**</sup>

\*  $p < 0.05$  Significant;  $p < 0.01$  \*\* highly Significant

c: compared between control and PCOS patients with child ;

N: compared between control and PCOS patients with no child;

M: compared PCOS patients with no child and with a child

FSH: follicle-stimulating-hormone ; LH: luteinizing-hormone ; PRL: prolactin- hormone

Table (2) shows that the elevation in the level (mean  $\pm$  SD) of LH hormone was more for the PCOS patients in the child group ( $7.8 \pm 3.2$ ) than the control group ( $5.4 \pm 2.1$ ), while the elevation was less for the PCOS patients with no child ( $5.4 \pm 2.5$ ), which is significantly increased ( $P < 0.01$ ) and this result was in line with a previous study [12].

The mean  $\pm$ SD of the LH/FSH ratio for PCOS patients with a child, with no child, and control groups, were ( $1.9 \pm 1.04$ ) ; ( $1.18 \pm 0.46$ ) and control ( $0.17 \pm 0.19$ ) group respectively with a significant increase ( $p < 0.01$ ) The increasing LH/FSH ratio is

a potent PCOS indication. And a dysfunctional hypothalamic-pituitary-ovarian axis could be the cause of the increased LH secretion. [16]

The activity and heightened pituitary response to GnRH, which in turn results in an increase in the LH/FSH ratio

The results mean  $\pm$ standard of the PRL for PCOS women with a child , PCOS women with no child, and control groups are ( $37.2 \pm 23.1$ ); ( $30 \pm 28$ ) and ( $18 \pm 6.3$ ) as shown in Table 2 respectively which is significantly increased ( $p < 0.01$ ) These results are in agreement with a previous study [17].

**Table (3):** Comparison between PCOS patients and controls groups according to the selected Hormonal profile in dependence on BMI

Parameter Groups	LH (mIU/ml) (mean ± SD)	FSH (mIU/ml) (mean±SD)	PRL (ng/m) (mean± SD)	Testosterone (ng/ml) (mean ± SD)	LH/FSH ratio (mean ± SD)
C1	5.014 ±1.71	7.33± 2.37	13.8± 4.22	3.14± 1.92	0.94± 0.38
C2	6.319 ± 2.584	4.22± 2.11	29.3± 7.28	5.405± 3.15	1.29 ± 0.575
G1	6.87 ± 3.455	5.04± 2.36	35.2± 24.9	4.207± 2.79	1.54± 0.69
G2	5.55± 1.935	7.68± 2.47	17.8 ±5.38	8.5± 0.577	1.17± 0.77
p- value c1α g1	0.03	0.000	0.000	0.000	0.02
p- value c2α g2	NS	0.000	0.03	0.000	0.000

C1. Control ≤ 25 Kg/m2 :C2.Control ≥25 Kg/m2 ,C1=35 C2=25

G1. Patients ≥ 25 Kg/m2:G2 Patients ≤25 Kg/m2 , G1=36 G2=24

FSH: follicle-stimulating-hormone ; LH: luteinizing-hormone ; PRL: prolactin- hormone

NS: Non-Significant

Table (3) shows that the elevation in the level (mean ± SD) of LH,FSH,PRL, Testosterone, and LH\FSH ratio hormone was PCOS patients in the (overweight, obese and normal weight women) , showed significant difference as compared to the (c1α g1 )(p<0.05 ) of LH , LH\FSH ratio while a highly significant increase (P<0.001) was found FSH,PRL,Testosterone, showed significant difference as compared to the ( c2α g2) on significant of LH hormone , showed significant difference as compared to the ( c2α g2) )(p<0.05 ) of PRL while a highly significant increase (P<0.001) was found FSH,Testosterone LH\FSH ratio , The results of our study were conducted with [18]

Weight gain is a big role in faulty and increased LH in charge of the corpus luteum and the leader, as there is influence on the body where the LH is developing at G1 and they have an increase or considerably while the G2 fell with natural weight and Weight gain is a big role in faulty and decreased FSH it is down And responsible for the follicle and the descent of the egg

The current study showed that level FSH is not frequently observed in patients PCOS comprising with control there was no significant difference in blood FSH levels between PCOS patients and the control group was consistent with the findings of the research [15]. Additionally, we noted increased LH levels and a lower LH / FSH ratio in PCOS individuals. control, although it has been stated about 75% of PCOS-afflicted females have increased LH levels. The pattern of GnRH stimulation heavily influences the production and secretion of LH and FSH. LH is favored by fast frequency, while FSH is favored by slower pulses. The primary factor causing this pattern of gonadotropin production is a faster GnRH pulse generator [19]

The current study showed that level PRL agreed with the results of S. Qian et al According to the Rotterdam criterion, patients with PCOS had considerably greater PRL levels than non-PCOS participants. One could argue that PCOS can be diagnosed with somewhat increased PRL levels, demonstrating its clinical importance. increased of the

present study’s findings[20] According to reports, gonadotropin-releasing hormone (GnRH)/LH pulse frequency is higher in PCOS-affected women (25), which may be the cause of elevated PRL levels.. [21] In GnRH neurons, many neurotransmitters and neuropeptide receptors have been identified that play a crucial role in the release of GnRH, LH, and FSH. Dopamine is one of these neurotransmitters that prevent LH from being released. PCOS is characterized by decreased dopaminergic tone, which is linked to increased LH secretion. Dopamine also prevents PRL from being released.. [22].

## Conclusion

In addition to insulin resistance, obesity, and impaired ovulatory function, PCOS is marked by severe metabolic abnormalities as well as underlying chronic low-grade inflammation. the syndrome of the poly cystic ovary can consider as a dis-order in the androgenic hormones, furthermore the gonadal atropine hormones.

## References

- N. Ajmal, S. Z. Khan, and R. Shaikh, "Polycystic ovary syndrome (PCOS) and genetic predisposition: A review article," *Eur. J. Obstet. Gynecol. Reprod. Biol.* X, vol. 3, p. 100060, 2019, doi: 10.1016/j.eurox.2019.100060.
- M. Yesiladali, M. G. K. Yazici, E. Attar, and F. Kelestimur, "Differentiating Polycystic Ovary Syndrome from Adrenal Disorders," *Diagnostics*, vol. 12, no. 9, pp. 1–14, 2022, doi: 10.3390/diagnostics12092045.
- S. H. Fatima, M. A. Naeem, A. Ali, and R. Asfar, "Prevalence of PCOS in Reproductive Age Women in Gujrat City," *EAS J. Radiol. Imaging Technol.*, vol. 4, no. 4, pp. 79–83, 2022, doi: 10.36349/easjrit.2022.v04i04.003.
- K.-T. Lu, Y.-C. Ho, C.-L. Chang, K.-C. Lan, C.-C. Wu, and Y.-T. Su, "Evaluation of Bodily Pain Associated with Polycystic Ovary Syndrome: A Review of Health-Related Quality of Life and Potential Risk Factors," *Biomedicines*, vol. 10, no. 12, p. 3197, 2022.

- H. M. Sadeghi et al., "Polycystic Ovary Syndrome: A Comprehensive Review of Pathogenesis, Management, and Drug Repurposing," *Int. J. Mol. Sci.*, vol. 23, no. 2, 2022, doi: 10.3390/ijms23020583.
- A. Bongrani et al., "High androgen concentrations in follicular fluid of polycystic ovary syndrome women," *Reprod. Biol. Endocrinol.*, vol. 20, no. 1, pp. 1–16, 2022, doi: 10.1186/s12958-022-00959-6.
- N. Scannell, L. Moran, E. Mantzioris, S. Cowan, and A. Villani, "Efficacy, Feasibility and Acceptability of a Mediterranean Diet Intervention on Hormonal, Metabolic and Anthropometric Measures in Overweight and Obese Women with Polycystic Ovary Syndrome: Study Protocol," *Metabolites*, vol. 12, no. 4, p. 311, Mar. 2022, doi: 10.3390/metabo12040311.
- N. Boisseau, "Physical Activity During the Perinatal Period: Guidelines for Interventions During the Perinatal Period from the French National College of Midwives," *J. Midwifery Womens. Health*, vol. 67, no. S1, Nov. 2022, doi: 10.1111/jmwh.13425.
- M. J. Khan, A. Ullah, and S. Basit, "Genetic basis of polycystic ovary syndrome (PCOS): Current perspectives," *Appl. Clin. Genet.*, vol. 12, pp. 249–260, 2019, doi: 10.2147/TACG.S200341.
- R. J. Swanson and B. Liu, "Conception and pregnancy," in *Fertility, Pregnancy, and Wellness*, Elsevier, 2022, pp. 53–71.
- S. Katta, "Urinary follicle-stimulating hormone glycoform analysis by automated western blot." Wichita State University, 2021.
- M. I. Borella, C. Chehade, F. G. Costa, L. zaro W. O. De Jesus, M. Cassel, and S. R. Batlouni, "The brain-pituitary-gonad axis and the gametogenesis," in *Biology and physiology of freshwater neotropical fish*, Elsevier, 2020, pp. 315–341.
- N. L. a Noob, "Old Account".
- S. H. H. Al-Musawy, I. E. Al-Saimary, and M. S. Flaifil, "Levels of cytokines profile in polycystic ovary syndrome," *Med. J. Babylon*, vol. 15, no. 2, p. 124, 2018.
- S. Qian et al., "Prolactin variants in human pituitaries and pituitary adenomas identified with two-dimensional gel electrophoresis and mass spectrometry," *Front. Endocrinol. (Lausanne)*, vol. 9, p. 468, 2018.
- N. Sarahian, H. Sarvazad, E. Sajadi, N. Rahnejat, and N. Eskandari-Roozbahani, "Investigation of common risk factors between polycystic ovary syndrome and Alzheimer's disease," 2021.
- K. Szosland, P. Paw owicz, and A. Lewi ski, "Prolactin secretion in polycystic ovary syndrome (PCOS)," *Neuroendocrinol. Lett.*, vol. 36, no. 1, pp. 53–58, 2015.
- I. M. Perovic Blagojevic et al., "Overweight and obesity in polycystic ovary syndrome: Association with inflammation, oxidative stress and dyslipidaemia," *Br. J. Nutr.*, vol. 128, no. 4, pp. 604–612, 2022, doi: 10.1017/S0007114521003585.
- R. Balasubramanian and W. F. Crowley Jr, "Isolated gonadotropin-releasing hormone (GnRH) deficiency," 2022.
- M. Saei Ghare Naz, M. Mousavi, F. Mahboobifard, A. Niknam, and F. Ramezani Tehrani, "A Meta-Analysis of Observational Studies on Prolactin Levels in Women with Polycystic Ovary Syndrome," *Diagnostics*, vol. 12, no. 12, p. 2924, Nov. 2022, doi: 10.3390/diagnostics12122924.
- A. Szeliga et al., "Neuroendocrine Determinants of Polycystic Ovary Syndrome," *Int. J. Environ. Res. Public Health*, vol. 19, no. 5, p. 3089, 2022.
- L. S. Mahmood, "Association of Zinc Alpha 2 Glycoproteins and Heat Shock Protein 70 with Polycystic Ovarian Syndrome in Iraqi Infertile Women . A Thesis," 1997.