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Deregulation of luteinizing hormone and testosterone levels in women with Polycystic Ovarian Syndrome: A descriptive analytical study

Duaa A H Al-Jubori[1], Alaauldeen S M AL-Sallami[2], Mohammad Alzeyadi[2]
[1] Department of Medical Laboratory Technics, Al-Mussaib Technical Institute, Al-Furat Al-Awsat Technical University, Babylon, Iraq
[2] Department of Biology, Collage of Science, University of Kufa, Najaf, Iraq

Abstract
Background: Polycystic ovarian syndrome (PCOS) is one of the most prevalent endocrine disorders in reproductive-age women, with prevalence of 10 –18 % according to diagnostic criteria.
Objective: The aim of this research is to determine the serum levels of LH and testosterone in women of reproductive age with PCOS.
Methods: This descriptive study design was conducted on ninety women, aged 18 to 45 years participate in this study, who were part of the study. Seventy of these women had polycystic ovarian syndrome, and the other 20 women were healthy as controls. Both were subdivided into two subgroups according to marital status (married and unmarried). Each participant underwent a clinical history, physical examination, and ovaries ultrasonogram. To assess the luteinizing hormone and testosterone levels, blood samples were collected from each participant, and enzyme linked immune sorbent assay (ELISA) technology was conducted after separating the serum.
Results: women with PCOS shows a significantly higher LH and testosterone levels compared with the control group.
Conclusion: Women with PCOS demonstrate distinct biochemical alterations, which include hormonal imbalances.

Keywords: Polycystic ovarian syndrome, luteinizing hormone, testosterone, ELISA

Introduction

Endocrine and metabolic disorders are prevalent among women of reproductive age, and Polycystic Ovarian Syndrome (PCOS) is one of them [1]. PCOS is a disorder that affacts the ovaries and may be associated with a number of clinical symptoms and hormonal disorders; therefore, PCOS is not one disease but, a group of related diseases together, which may lead the ultimately lead to infertility resulting from chronic anovulation. Some cases of PCOS do not present with obvious symptoms except several small cysts in size in the ovaries, which can be observed during vaginal examination by ultrasound [2], but in most cases, this syndrome is accompanied by several clinical symptoms, which can be observed in PCOS, such as menstrual cycle disorders (amenorrhea or oligomenorrhoea), anovulation, infertility, hirsutism, acne, and obesity (9–11) and these symptoms are often caused by hyperandrogenism caused by insulin resistance and hyperinsulinemia [3].

The diagnosis of PCOS involves the assessment of clinical symptoms, such as irregular menstrual cycles and signs of androgen excess, as well as measuring hormonal levels and detecting ovarian cysts using ultrasound. Luteinizing hormone (LH) excess is common and necessary for the expression of gonadal steroidogenic enzymes and sex hormone secretion, but is less likely to be the primary cause of ovarian androgen excess because of LH-induced desensitization of theca cells [4]. Testosterone is one of the androgens; the major circulating androgens in women are testosterone, dihydrotestosterone, dihydroepiandrosteendione (DHEA) and Dihydroepiandrostenodione – sulphate (DHEA-s), both the adrenal gland and ovaries normally synthesize and secret testosterone, and the other androgens approximately 50% of testosterone arises from the peripheral conversion of androstendione, and 25% is secreted by the adrenal gland [5]. Hypersecretion of Luteinizing Hormone (LH) is a prominent feature of the deregulation occurring in hypothalamic-pituitary-ovarian (HPO) axis which is a central component in PCOS pathogenesis. This neuroendocrine dysfunction is reflected by increased both frequency and amplitude of Gonadotropin-Releasing Hormone (GnRH) pulses, favoring LH production in the pituitary gland over Follicle-Stimulating Hormone (FSH) [6]. This increases the LH:FSH ratio, which is an important factor for increased androgen production in the ovary. LH binds to Luteinizing hormone receptors (LHCGR) present on ovarian theca cells and exerts its action, upregulating the expression of important steroidogenic enzymes in these cells like CYP11A1 and CYP17A1 that stimulate increases in the production of androstenedione and testosterone [7].

In addition, hyperinsulinemia has a synergic contribution to this hormonal deregulation. Insulin has a co-gonadotropin action on the theca cell, increasing its responsiveness to LH, and also it inhibits the hepatic synthesis of Sex Hormone-Binding Globulin (SHBG). The lowering of the SHBG causes increased levels of free, biologically active testosterone which worsens the clinical features of hyperandrogenism [8]. Recent research has also identified that Chronic low-grade inflammation further sensitizes theca cells to LH stimulation and thus there is a complex interaction between metabolism and endocrinology [9]. In the genetic context, variants of the gene DENND1A have been strongly associated with this deregulation, it favoring the intrinsic increase in androgen production of theca cells independent of gonadotropin levels [10]. Further, Anti-Müllerian Hormone (AMH) has become a major focus as hyperamhostatism in PCOS has been shown to suppress the effect of FSH on aromatase, which impairs the conversion

of androgens to estrogens, and to feed back to the hypothalamus to increase the frequency of GnRH pulses [11]. The abilities to grasp these complex processes of hormonal deregulation are important in the analysis of the results of the clinical study carried out on LH and testosterone deregulations in affected women [12]. Study has indicated that a higher baseline LH and testosterone levels in PCOS-affected women were linked to worse ovulatory responses; thus, the aim of this study is to assess the serum LH and testosterone levels in patients with PCOS and compare the levels with healthy women in AL-Hilla City.

Methodology

Subject

Ninety women, ranging in age from 18 to 45, who had not reached menopausal, participated in the present categories in two groups; 70 women with polycystic ovary syndrome (PCOS) were performed at the private clinic for obstetrics and gynecology in AL-Hilla city, Iraq and diagnosed with PCOS by physicians according to the Rotterdam classification (2003) and divided into two subgroups according to marital status (35 married and 35 unmarried), and 20 healthy women had regular menstruation periods, with normal ovaries as observed by the gynecologists. They were divided into two subgroups according to marital status (10 married and 10 unmarried). All patients and control were subjected to Ultra-sonography for diagnosis of both ovaries.

Data collection

Data were collected from March to July, 2023. Clinical information was collected for each patient in the PCOS group using a carefully designed questionnaire. The questionnaire for the control group was the age, weight and height, hip, and waist circumferences and menstrual regularity with fertility and hirsutism. Not smokers with no kidney disease, liver disease, cancer, strokes, any acute or chronic inflammatory disease, cerebrovascular accidents, alcoholics, rheumatoid arthritis, autoimmune disease, patients with type 1 and 2 diabetes mellitus, and no history of contraceptive drugs.

Exclusion criteria

Women who suffered from diseases (autoimmune disease, diabetes mellitus, thyroid disease, cardiovascular disease, hypertension, chronic renal failure, and malignant diseases) were excluded, and those who had a history of receiving any other medication (lipid reduction, ovulation stimulation, corticosteroids, antidiabetic, and antihypertensive medications) within 6 months were also excluded.

Sample collection and preparation

5 mL of venous blood was withdrawn by a medical syringe on the follicular phase (2-5) day of the menstrual cycle of each selected subject (patients and controls). Each woman has a special form written on it with the name, date, and serial number. Blood Samples were processed within one hour after collection, and then placed in a centrifuge at 3000 RPM for 20 minutes to separate the serum sample. The serum was transferred into plastic cuvettes, which was used for measuring hormones, to be frozen at -20 °C until use, when necessary.

Hormone level measurements

Blood serum samples were analyzed to determine the levels of luteinizing hormone (LH) and testosterone. Before analysis, serum samples were equilibrated at room temperature. Hormone concentrations were measured using commercial enzyme-linked immunosorbent assay (ELISA) kits (Demeditec Diagnostics GmbH, Germany). Individual assays were performed according to the manufacturer's instructions using sandwich ELISA technology

Statistical Analysis

Data were analyzed using SPSS software (Version 21). Standard Error (SE) and (mean ±SE) were used to describe continuous data. The t-test and Duncan-tests were used to compare variables between the total control number and the number of patients according to the p-value (p-value less than 0.05 was considered significant).

Results

In this study, the cases (PCOS patients) and controls were comparable (similar) in age, 28.34 years versus 28.95 years, as shown in (Table 1).

Table 1: Mean Age Distribution between the studied groups

Characteristics	PCOS group (n=70)	Control group (n=20)	p-value
Age (year)	28.34 ± 0.82	28.95 ± 1.02	N.S.
NS: Non significant			

Data are presented as mean ± SE, SE; Standard Error PCOS, Polycystic Ovary Syndrome

(Table 2) Highlights the mean age distribution in two groups (married and unmarried) of women with PCOS and control. A comparable age distribution was found between PCOS patients and control participants in both married and unmarried categories, with no statistically significant differences observed among the groups, indicating that age was similar across all study groups.

Table 2: Comparison of age distribution among married and unmarried women with PCOS and control groups

Characteristics Mean (yr.) ±SE	PCOS group		control group		p-value
	Married (n=35)	Unmarried (n=35)	Married (n=10)	Unmarried (n=10)	
Age	33.11 ± 0.90	23.57 ± 0.79	32.00 ± 1.05	25.90 ± 1.12	N.S.

N.S. = Not Significant p> 0.05

(Table 3) presents the comparison of the mean value of the selected hormonal profile between patients with PCOS and controls. The results show a highly statistically significant increase in the PCOs Phenotypes in compared with the mean of the control group regarding HL and testosterone levels.

Table 3: Comparison of serum LH and testosterone levels between PCOS patient and controls

Hormone Concentration	PCOS group (n=70)	Control group (n=20)	p-value
	Mean ± SE	Mean ± SE	
LH (ng/mL)	7.21±0.71	3.19±0.57	0.01**
Testosterone (ng/mL)	0.82± 0.05	0.41±0.05	0.01**

Luteinizing Hormone (LH), ** Significant difference at $p \leq 0.01$

(Table 4) shows no statistically significant differences (p>0.05) in serum luteinizing hormone (LH) levels among married and unmarried participants in both PCOS patient

and control groups. Similarly, testosterone levels did not show statistically significant differences between groups, despite an observed higher mean value in married patients compared with other groups. Overall, variations in both LH and testosterone levels across the studied groups were non-significant.

Table 4: Luteinizing hormone and testosterone levels of cases and controls between married and unmarried subjects

Characteristics (Mean ± SE)	PCOS group		Control group		p-value
	Married (n=35)	Unmarried (n=35)	Married (n=10)	Unmarried (n=10)	
LH (ng/mL)	6.31 ± 1.07	8.12 ± 0.92	3.73 ± 0.92	6.65 ± 0.70	**N.S.**
Testosterone (ng/mL)	1.02 ± 0.05	0.62 ± 0.08	0.32 ± 0.05	0.50 ± 0.10	**N.S.**

N.S. = Not Significant p>0.05

Discussion

PCOS still does not have a clear cause, and subjective phenotypes make it difficult to make a complete diagnosis. PCOS is the most common hormonal disorder among women of reproductive age. Mean age of patients and control in this study was approximately 28 years. It is evident that the two groups are approximately well matched; thus, the results obtained could be considered creditable, which is in line with the results of other studies [13]. As this endocrinological problem is common among women of reproductive age, early diagnosis and management are useful to prevent short-term as well as long-term sequelae of the disease. Elevated LH is a common feature of polycystic ovarian syndrome ; however, it is not required for diagnosis. A discernible change in serum levels between the two groups was observed, and both were significantly effective in increasing the serum levels of LH and testosterone in individuals suffering from PCOS.

Luteinizing hormone (LH) is an essential component of ovarian physiology, stimulating ovulation, luteinization, and androgen production. LH promotes androgen production mainly via binding to LH receptors in ovarian theca cells, which further increase – among others – testosterone synthesis [14]. Increased LH levels are associated with severe forms of the PCOS phenotype by multiple studies. Excessive LH stimulation interrupts normal follicular development, leading to the accumulation of multiple follicles that are held in developmental arrest at the preantral and antral stages. This condition causes excessive theca cell hyperplasia and accumulation of follicular fluid, leading to cyst formation along the periphery of the ovary that leads to the characteristic 'string of pearls' appearance seen in PCOS. In addition, enhanced LH pulse frequency can compromise gonadotropin secretion balance by decreasing relative FSH production and modifying estrogen synthesis. Such a phenomenon results in an imbalance between reproductive hormones that slows the process of follicular maturity and prevents ovulation, leading to polycystic ovaries [15]. However, baseline levels of LH are normal in some women with PCOS. Such heterogeneity can be attributed to different phenotypes of PCOS, including typical and atypical forms of this syndrome, along with differences in LH pulsatility and episodic secretion patterns that are not always detected by single base-line measurements [16].

Our findings differ from those reported in a previous study conducted among Iraqi women, which documented a statistically significant elevation of LH levels in patients with PCOS compared with controls**.** As with the current study, the findings of previous studies demonstrated higher mean LH concentrations in patients with PCOScompared with controls [17-19]. Such discrepancies may be attributed to differences in sample size, study population characteristics, or methodological variations between studies. The present study demonstrated significantly higher testosterone levels in women with PCOS compared with healthy controls. This can be explained by the alteration of hypothalamic–pituitary–ovarian axis activity, which results in increased pulsatile secretion of GnRH and preferential stimulation of LH release from the pituitary gland. Elevated LH subsequently stimulates ovarian theca cells, leading to increased androgen production, particularly testosterone. Consequently, the higher testosterone levels observed in the PCOS patients compared with the control group are consistent with the hyperandrogenic state that characterizes this disorder [20].

The present findings are consistent with several international studies, including those by Bereshchenko *et al.*, [21], which reported comparable results. In addition, similar observations have been documented in Iraqi populations, as demonstrated in the study conducted by Ibrahim *et al.,* AL-Tikrit *et al.,* and Sharif *et al.,* 2017 [22-24]. As seen in the earlier results indicating increased hormonal levels among patients with PCOS compared to controls, the current findings show variability in levels of LH and testosterone among the groups studied. The PCOS group had higher mean levels, but they were not significant (p>0.05). The lack of significance might be related to sample size and the presence of individual variation over hormonal levels.

Conclusion

The present study concludes that LH and testosterone are higher in polycystic ovary syndrome (PCOS) patients than in healthy women. These findings emphasize the need for comprehensive monitoring in patients with PCOS to mitigate associated risks and plan therapeutic interventions accordingly

Declarations

Funding source

The authors received no funding from any source.

Limitations

We can conclude that our study has a number of limitations. The sample size in each group was small and our findings require confirmation in larger studies.

Ethical approval

The study was conducted following the ethical principles of the before the sample was collected, written informed consent was obtained from all participants prior to sample collection. The University of kufa, college of Science, Biology department, reviewed and approved the study protocol, and subject information.

References

1. Singh S, Pal N, Shubham S, *et al*. Polycystic ovary syndrome: etiology, current management, and future

therapeutics. Journal of Clinical Medicine,2023:12(4):1454.
2. Naeem I, Zehra A, Rehman F, *et al*. Polycystic ovarian syndrome a risk factor for non-communicable diseases: insights into recent research and prevention approaches. Journal of Ovarian Research,2025:18(1):1-15.
3. Witchel SF, Oberfield SE, Peña AS. Polycystic ovary syndrome: pathophysiology, presentation, and treatment with emphasis on adolescent girls. Journal of the Endocrine Society,2019:3(8):1545-1573.
4. Mansour A, Noori M, Hakemi MS, *et al*. Hyperandrogenism and anthropometric parameters in women with polycystic ovary syndrome. BMC Endocrine Disorders,2024:24(1):201.
5. Motlagh Asghari K, Nejadghaderi SA, Alizadeh M, *et al*. Burden of polycystic ovary syndrome in the Middle East and North Africa region, 1990-2019. Scientific Reports,2022:12(1):7039.
6. Satapathy P, Vadia N, Menon SV, *et al*. Prevalence of polycystic ovarian syndrome in South Asian countries and 21 global regions with sociodemographic index: a systematic analysis of the global burden of disease (1990-2021). Journal of Gynecology Obstetrics and Human Reproduction,2025:54:103019.
7. Teede HJ, Tay CT, Laven JS, *et al*. Recommendations from the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome. The Lancet Diabetes and Endocrinology,2023:11(12):954-970.
8. Azziz R, Carmina E, Dewailly D, *et al*. The androgen excess and polycystic ovary syndrome society criteria for the polycystic ovary syndrome: the complete task force report. Fertility and Sterility,2009:91(2):456-488.
9. Rudnicka E, Suchta K, Grymowicz M, *et al*. Chronic low grade inflammation in pathogenesis of PCOS. International Journal of Molecular Sciences,2021:22(7):3789.
10. Ham KVD, Laven JS, Tay CT, *et al*. Anti-müllerian hormone as a diagnostic biomarker for polycystic ovary syndrome and polycystic ovarian morphology: a systematic review and meta-analysis. Fertility and Sterility,2024:122(4):727-739.
11. McAllister JM, Legro RS, Modi BP, Strauss JF. Genomic and proteomic evidence for hormonal and metabolic dysregulation in PCOS: an update. Nature Genetics, 2025. In press.
12. Escobedo M, Taylor HS. Polycystic ovary syndrome: an update on diagnosis and management. Cleveland Clinic Journal of Medicine,2026:93(3):176-185.
13. Witchel SF, Burghard AC, Tao RX, Oberfield SE. The heterogeneity of polycystic ovary syndrome: a focus on hormonal deregulation. Frontiers in Endocrinology,2022:13:717472.
14. Oduwole OO, Huhtaniemi IT, Misrahi M. The roles of luteinizing hormone, follicle-stimulating hormone and testosterone in spermatogenesis and folliculogenesis revisited. International Journal of Molecular Sciences,2021:22(23):12735.
15. Alani WTAL. The genetic polymorphism in vitamin D and insulin receptors gene in association with polycystic ovarian syndrome [doctoral dissertation]. University of Anbar, 2025.
16. Saadia Z. Follicle stimulating hormone (LH:FSH) ratio in polycystic ovary syndrome (PCOS)-obese vs. non-obese women. Medical Archives,2020:74(4):289.
17. Al-Jebori SR. Effect of physical activity on sex hormones in polycystic ovary syndrome Iraqi women. Iraqi Journal of Biotechnology,2018:17(1):98-105.
18. Abdulwahid H, Hussein B, Omran Z, Alhasanawy S. Disorders of sex hormones and lipid profile in obese and non-obese women with polycystic ovary syndrome (PCOS) in Karbala City. Annals of Tropical Medicine and Public Health,2019:22:100-106.
19. Al-Jawadi ZA, Al-Taie FK. Clinical study of the role of thyroid stimulating hormone on polycystic ovary syndrome. Thyroid,2025:7:8.
20. Razzaq T, Arif M, Ahmad SZ, Shabbir H. The pathogenesis of polycystic ovary syndrome (PCOS): the hypothesis of functional ovarian hyperandrogenism revisited. Contemporary Journal of Social Science Review,2025:3(1):1808-1822.
21. Bereshchenko O, Bruscoli S, Riccardi C. Glucocorticoids, sex hormones, and immunity. Frontiers in Immunology,2018:9:1332.
22. Ibrahim MII, Al-Saffar JM. Serum level evaluation of interleukin-18 in obese women with polycystic ovary syndrome. Iraqi Journal of Science,2018:59:1989-1994.
23. Al-Tikriti SB, Naji NA. Estimation of the hepcidin level and some biochemical parameters in patients of polycystic ovary syndrome in Kirkuk city. Tikrit Journal of Pure Sciences,2019:24(4):34-39.
24. Sharif E, Rahman S, Zia Y, Rizk NM. The frequency of polycystic ovary syndrome in young reproductive females in Qatar. International Journal of Women's Health,2017:9:1-10.