IRAQI Academic Scientific Journals
Samarra Journal of Pure and Applied Science
SJPAS
مجلة سامراء للعلوم الصرفة والتطبيقية
www.sjpas.com
p ISSN: 2663-7405
e ISSN: 2789-6838

A Clinical Study of Hormonal Profile Alterations in Women with Polycystic Ovary Syndrome in Samarra City

Maysam Ibrahim Mahdi[1*], Reem Ibrahim Mahdi [2], Ayat Muwafaq Majeed[1] , Zainab Hussain Ali [2]
1 Department of Biology, College of Education for Pure Science, University of Samarra, Salah Aden, Iraq.
2 Department of Chemistry, College of Education for Pure Science, University of Samarra, Salah Aden, Iraq.

https://doi.org/10.54153/sjpas.2025.v7i4.1272

Article Information
Received: 28/05/2025
Revised: 30/06/2025
Accepted: 01/07/2025
Published: 30/12/2025

Keywords:
Polycystic Ovary Syndrome, sexual hormones, prolactin, Anti-mullerian hormones.

Corresponding Author

E-mail: maysam@uosamarra.edu.iq
Mobile:

Abstract

This study evaluated the polycystic ovary syndrome affection on the levels of selected sexual hormones, including luteinizing hormone, follicle-stimulating hormone, estrogen, progesterone, and other related hormones such as anti-Mullerian hormone, and prolactin, among a sample of women from Samarra, Iraq. The study was conducted by selecting 40 women, 15 were a healthy woman which represented the control group and, 25 were represented the patients group after they were diagnosed with polycystic ovary syndrome by a specialist doctor and underwent the necessary examinations and ultrasound during the period from October 2024 to January 2025. Hormone levels were measured using serum tests, and the data were analyzed using an independent t-test using SPSS version 25. The results of this study showed that the group of women with polycystic ovary syndrome had significantly higher levels of luteinizing hormone, estrogen anti-Müllerian hormone, and prolactin, and significantly lower levels of follicle-stimulating hormone and progesterone compared to the control group ($p \leq 0.005$).

Introduction

Polycystic cystic Ovary Syndrome (PCOS) was first described in 1935 and was originally named Stein-Leventhal Syndrome. Other historical terms used to describe the condition include functional ovarian hyperandrogenism, hyperthecosis, and sclerocystic ovary syndrome [1], it affects women in age (12–45 years) and it is one of the causes of female subfertility and the most common endocrine disorder among women in reproductive age [2]. The condition may be present from birth but typically does not manifest symptoms until puberty. The clinical features of this disorder may change throughout the lifespan, from adolescence to menopause. However, no significant effort has been made to characterize age-related variations in phenotype and clinical presentation [3]. There are several factors that cause PCOS; Hyperinsulinemia which refers to the resistance of insulin that requires abnormally high amounts of insulin to stimulate a cellular response and is the commonly clinical symptom in women with the syndrome, it is increases the effect of luteinizing

hormone (LH) in which leading to increased production of androgens [4]. Also, Insulin resistance may be an important first indicator of metabolic disorders in women at risk for metabolic syndrome and coronary artery disease, particularly after a diagnosis of PCOS is confirmed [5]. As for the effect of hyperandrogenism on PCOS, the main source of androgen excess in PCOS is ovary, due to high levels of LH, as ovarian dysfunction leads to a decrease in its sensitivity to this hormone [6]. The elevation in LH levels is a result of disruption in the hypothalamic-pituitary-gonadal axis. Furthermore, hyperandrogenemia weakens the ability of progesterone to inhibit Gonadotropin-releasing hormone (GnRH) [7]. Thus, elevated GnRH levels lead to an increase in LH secretion and a decrease in Follicle-Stimulating Hormone (FSH), which converts androgens to estrogen. Therefore, elevated ovarian androgens impede follicle maturation and prevent the transition from an androgen-dominated environment to an estrogen-dominated environment, which is necessary for normal follicular development. This results in polycystic ovaries, a hallmark of PCOS [8, 9].

The same applies to hormones, which are considered a chemical substance secreted by endocrine glands within the body in small quantities, some are composed of proteins, some of lipids, or other compounds. Some hormones require specific plasma proteins to facilitate their transport through the bloodstream to the target organ or tissue. Thus, hormones affect the target organ to perform a specific function [10]. They affect the target tissues to stimulate chemical changes that lead to a response from the relevant organ [11]. Among the hormones related to PCOS are: LH, FSH, Estrogen (E2), Progesterone, Anti mullerian Hormone (AMH) and, Prolactin Hormone (PRL). luteinizing hormone also known as lutropin, is a glycoprotein and also includes carbohydrate components, which account for approximately 20% of its molecular weight [12]. LH is secreted by the gonadotropic cells of the anterior pituitary gland and regulated by the hypothalamus. luteinizing hormone stimulates the production of steroid hormones testosterone in males and estradiol in females and, in conjunction with FSH, stimulates the growth of ovarian follicles, which enhances steroid hormone production and determines the timing of ovulation. [13]. follicle-stimulating hormone is a glycoprotein composed of alpha and beta subunits; the alpha is shared with LH, while the beta confers biological specificity [14]. It is secreted by anterior pituitary gonadotrophs under hypothalamic control. In women FSH acts synergistically with LH to promote follicular development and steroid hormone production, with secretion peaking mid-cycle around ovulation. During menopause, reduced inhibin leads to elevated FSH levels, combined measurement of FSH and LH is essential for evaluating reproductive function [15]. Estrogen is primarily in the form of estradiol, the primary hormone produced by the ovary, while estrone and estriol are largely peripheral metabolites [16].

Estradiol is secreted from the ovarian follicles during the menstrual cycle and from the placenta during pregnancy. Its receptors are found in multiple tissues such as bone, breast, and liver. Synthesis and secretion of estrogen are regulated from the hypothalamic-pituitary axis by the hormones LH and FSH. Estradiol levels are commonly assessed along with LH, FSH, and progesterone when investigating infertility. At low estradiol levels, LH and FSH secretion is slightly inhibited, while at high levels, they are more strongly inhibited. Peripheral estrone production may also be increased in cases of mild obesity or in disorders such as PCOS [17]. As for the role of progesterone, it is a steroid hormone synthesized from cholesterol via enzymatic steps involving cytochrome P450scc (CYP11A1) and 3β-hydroxysteroid

dehydrogenase [18]. It is a precursor for other steroid hormones, such as cortisol, aldosterone, testosterone, and estradiol. At first Progesterone is produced by the corpus luteum during the luteal phase of the menstrual cycle and by the placenta starting in the eighth week of pregnancy. Small amounts are also secreted by the adrenal glands, particularly during stress, and by the central nervous system, where it acts as a neurosteroid [19]. Progesterone role an important function in regulating the menstrual cycle, preparing the endometrium for implantation, and maintaining pregnancy by inhibiting uterine contractions [20]. And the other related hormones are AMH and PRL, AMH is a glycoprotein belonging to the transforming growth factor-beta (TGF-β) family, composed of two identical subunits linked by disulfide bridges, with a molecular weight of approximately 140 kDa [21].

Anti mullerian Hormone is secreted by Sertoli cells in males, during fetal development to inhibit the formation of Müllerian ducts. In females, it is produced by granulosa cells of preantral and small antral follicles, where it plays a key role in regulating folliculogenesis by inhibiting primordial follicle recruitment and reducing follicular sensitivity to FSH, thereby preserving ovarian reserve [22]. While (PRL) is a glycoprotein hormone composed of 198 amino acids and exists in monomeric and dimeric forms. It is synthesized by the anterior pituitary gland, with its primary physiological role in females being the initiation and maintenance of lactation, in addition to its involvement in follicular maturation and oocyte development [23]. Elevated PRL levels can suppress estrogen in women and testosterone in men, approximately 15–20% of women with the syndrome also exhibit hyperprolactinemia [24].

The current study aimed to**:** Compare LH level between women with syndrome and healthy women, and determine its significance in the development of the syndrome. Evaluate FSH levels and their role in influencing ovarian function in PCOS patients, and Evaluate AMH concentrations as a potential biomarker of ovarian reserve in women with PCOS compared to healthy women by Measure prolactin PRL levels in both groups and evaluate their physiological significance in influencing or not influencing the development of the syndrome.

Materials and methods

Experimental Design

This study was conducted in selected medical laboratories affiliated with gynecology clinics in Samarra from October 14, 2024, to January 20, 2025. It included 25 women aged 22–43 years diagnosed with PCOS based on clinical and medical evaluations by specialists. A control group of 15 healthy women was also enrolled, confirmed to be free of (PCOS), hormonal disorders, or chronic diseases through clinical assessment and laboratory testing included the evaluation of sex hormone levels, including (LH, FSH), estrogen, and progesterone, and also included the assessment of AMH and PRL levels. They were measured during the early follicular phase of the menstrual cycle, specifically between days 2 and 5 from the onset of menstruation.

Collection of blood sample

Blood samples were collected using 5 ml syringes and placed in gel tubes. The samples were left at room temperature for 15 minutes, then centrifuged at 3000 rpm for 15 minutes to obtain the serum. The serum was subsequently stored at -20°C until hormonal assays were performed.

Statistical analysis

The collected data were analyzed using appropriate statistical methods. Descriptive statistics, including means and standard deviations (SD), were calculated for all variables. To assess differences between groups, an independent t-test was used, with a significance level set at $p \leq 0.05$. All statistical analyses were performed using SPSS version 25.

Results and Discussion

The hormone levels of the study groups were assessed to confirm the samples of both the control and the patient groups. The results are presented in Table 1:

Table 1: The mean±SD of Hormones levels between the control and the patient groups

Hormones	Control	Patients	P value
LH IU/L	4±0.71 b	9.06±0.51 a	0.0001
FSH IU/L	7.2±1.27 a	4.08±0.44 b	0.0001
Estrogen pg/mL	30.45±2.69 b	58±2.73 a	0.0001
Progesterone ng/mL	2.08±0.09 a	0.28±0.07 b	0.005
Anti mullerian ng/mL	1.53±0.82 b	4.28±0.73 a	0.0001
Prolactin IU/L	12.18±0.33 b	18.41±3.77 a	0.0001

Luteinizing Hormone Level

The mean ± ± S.D of LH levels in the control group was 4 ± 0.71 IU/L, while in the patient group, it was 9.06 ± 0.51 IU/L, as shown in Figure 1.

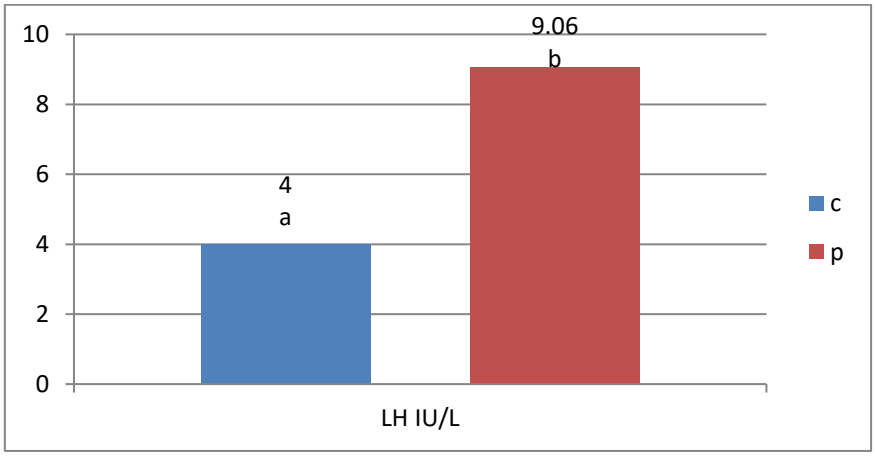

Figure 1: Luteinizing Hormone Level.

The results showed a significant increase in LH levels in women with PCOS compared to healthy women ($p \leq 0.005$). This is consistent with the study [24], which found that 88% of women with PCOS had elevated LH levels [25], also observed a correlation between LH levels and gonadotropin receptors in cases of oligomenorrhea. High LH levels and elevated androgens disrupt normal ovarian function [26]. In PCOS low to normal FSH and high LH prevent the LH surge necessary for ovulation, resulting in irregular cycles. Additionally, the imbalance between LH and FSH stimulates inappropriate testosterone production, interfering with follicle growth and ovulation [27].

Follicle Stimulating Hormone level

The mean ± S.D of the FSH levels in the control group was 7.2 ± 1.27 IU/L, while in the patient group, it was 4.08 ± 0.44 IU/L, as shown in Figure 2:

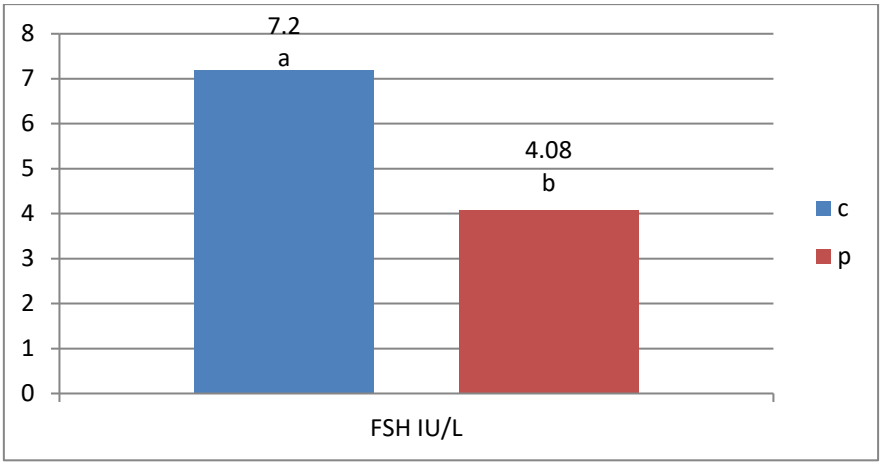

Figure 2: Follicle Stimulating Hormone Level.

The results showed a significant decrease in FSH levels in the patient group compared to the healthy group ($p \leq 0.005$). These findings align with [28], who indicated that women with PCOS experience disruptions in the HPO axis, which leading to an increased in LH/FSH ratio, elevated androgen levels, and reduced FSH. This hormonal imbalance prevents proper follicular maturation, causing polycystic ovaries. The decrease in FSH in PCOS is due to a higher LH secretion driven by an increased pulsatile release of GnRH from the hypothalamus, as a result the pituitary stimulating to secrete more LH than FSH. This disturbance leads to increased androgen production in the ovaries, inhibiting follicular maturation and preventing ovulation [29].

Estrogen hormone level

The mean ± S.D for estrogen levels in the control group was 30.45 ± 2.69 pg/ml, while in the patient group it was 58 ± 2.73 pg/ml, as shown in figure 3.

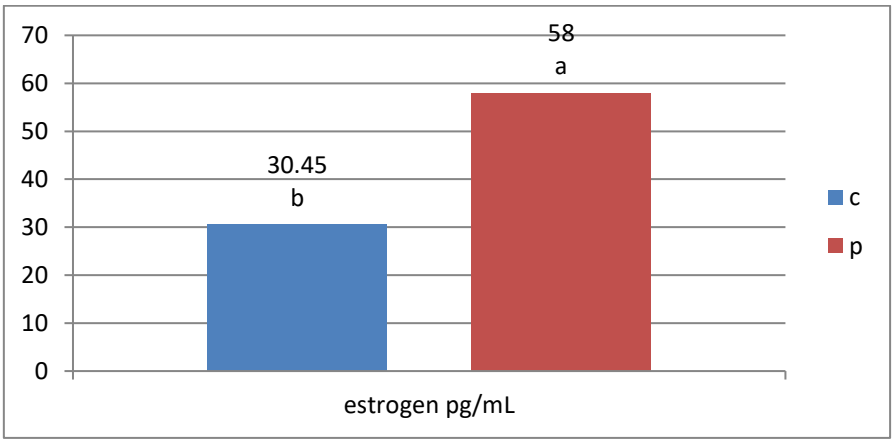

Figure 3: Estrogen hormone level.

The results showed a significant increase in estrogen levels in the patient group compared to the control group. This aligns study [30], which indicated that disruptions in estrogen signaling and receptors (ERα and ERβ) contribute to PCOS development. The study explained

that estrogen regulates follicular growth, and its disruption leads to arrested growth and estrogen accumulation. Continuous stimulation of immature follicles results in low FSH levels, preventing full maturation and leading to estrogen buildup. Additionally, lack of ovulation causes elevated estrogen levels and insufficient progesterone, which can lead to endometrial thickening and irregular bleeding [31]. Some women show a condition known as estrogen dominance, where the estrogen level higher-than-normal levels. This can occur in women with (PCOS), this hight levels can cause irregular menstrual cycles, unwanted hair growth, and acne [32].

Progesterone hormone level

The mean ± S.D of progesterone levels in the control group was 2.08 ± 0.09 ng/mL, while in the patient group it was 0.28 ± 0.07 ng/mL, as shown in Figure 4.

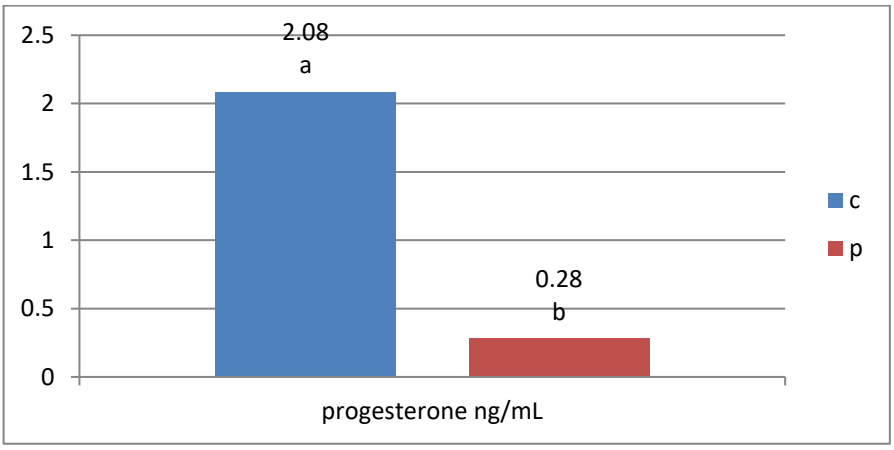

Figure 4: Progesterone hormone level.

The results showed a significant decrease in progesterone levels in the patient group compared to the healthy group. These findings are consistent with the study [33], which indicated that PCOS is associated with increased LH pulse frequency, ovarian dysfunction, and increased androgen production, leading to lower progesterone levels. Additionally, the study [34] highlighted that women with the syndrome exhibit reduced expression of enzymes responsible for progesterone synthesis, such as CYP11A1 and HSD3B2, which results in reduced progesterone production and disrupts follicular development and ovulation. The decrease in progesterone in PCOS is mainly due to anovulation or irregular ovulation, that lead to impairs the formation of the corpus luteum which responsible for progesterone production in the second half of the menstrual cycle. This reduction in progesterone contributes to menstrual disturbances, difficulty in conception, and increased risk of endometrial hyperplasia [35].

Anti-Mullerian Hormone level

The mean ± S.D. of AMH levels was 1.53 ± 0.82 ng/mL in the healthy group, whereas it was 4.28 ± 0.73 ng/mL in the control group, as illustrated in Figure 5.

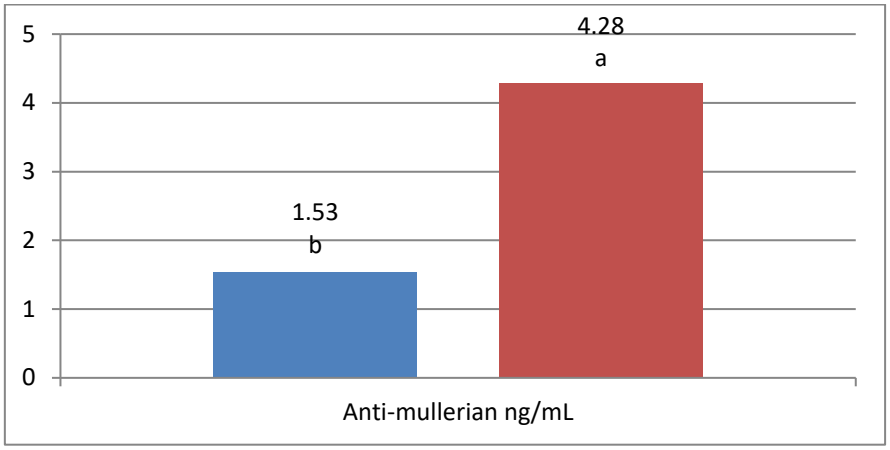

Figure 5: Anti-Mullerian Hormone Level.

The results showed a significant increase in AMH levels in the patient group compared to healthy controls (P ≤ 0.005). This is consistent with [36], who showed higher AMH levels in women with syndrome and a positive association between AMH and the number of small follicles, supporting its diagnostic value. Similarly [37], reported that AMH levels were 7.84 ± 3.67 ng/mL in PCOS patients versus 3.23 ± 1.56 ng/mL in healthy women, and showed a positive association with ovarian volume and follicle count. Anti-Mullerian hormone level is markedly elevated in women with PCOS, representing a key biochemical feature of the disorder. This increase is mainly attributed to a higher number of small antral follicles (2–9 mm) in the ovaries, which produce more AMH. Studies have shown that each follicle in PCOS ovaries secretes greater amounts of AMH compared to those in healthy ovaries. Additionally, elevated AMH levels may inhibit follicular growth and maturation, contributing to the ovulatory dysfunction commonly associated with PCOS [38].

Prolactin hormone level

The mean ± S.D. of serum Prolactin levels was 12.18 ± 0.33 IU/L in the control group and 18.41 ± 3.77 IU/I in the patient group, as illustrated in Figure (6).

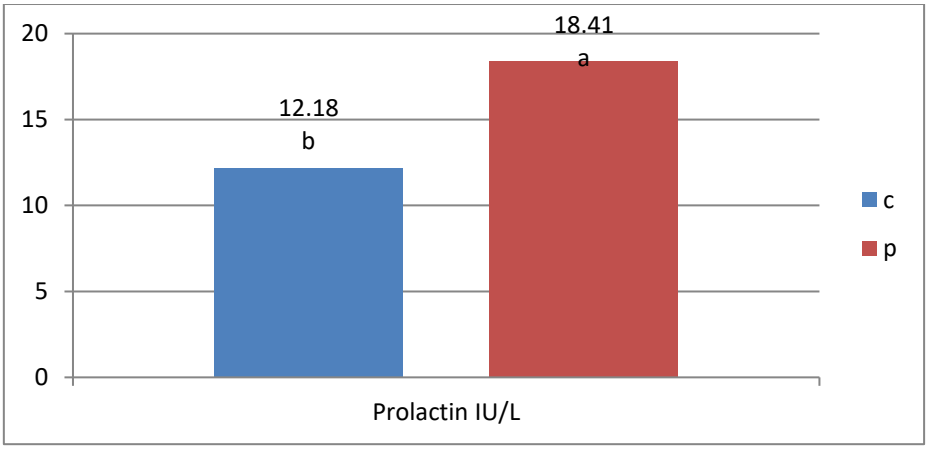

Figure 6: Prolactin hormone Level.

The results showed a significant increase in serum prolactin levels in the patient group compared to the healthy controls (P ≤ 0.005). These findings align with [39], who found slightly elevated prolactin levels in women with PCOS, although this increase was not clinically significant. Similarly, as [40], observed that 69.4% of women with PCOS had elevated

prolactin levels (mean 31.17 ± 10.24 ng/dL), which were associated with lower pregnancy rates, suggesting a negative impact on fertility.

However, the results differ from those of [41], who reported lower prolactin levels in women with PCOS and identified correlations with metabolic markers like glucose and lipids, indicating a potential link to metabolic risks in PCOS. The increase in prolactin in PCOS may be due to increased GnRH pulse frequency, reduced dopaminergic inhibition, and relative estrogen excess, which stimulates prolactin secretion [42,43].

Conclusion

The results confirm that polycystic ovary syndrome PCOS is associated with a clear hormonal disorder that may contribute to ovulation disturbances and decreased fertility. The overall hormonal profile revealed by the study indicated a multifactorial endocrine disorder in PCOS cases. These findings highlight the importance of early hormonal screening in suspected cases to ensure timely diagnosis and intervention.

References

1- Group, T. E. A. R. E. W., Fauser, B. C. J. M., Diedrich, K., Bouchard, P., Dominguez, F., Matzuk, M., ... & Howles, C. M. (2011). Contemporary genetic technologies and female reproduction. *Human reproduction update*, *17*(6), 829..

2- Legro,RS. 2017. Stein-Leventhal syndrome. In: *Encyclopedia Britannica* [Internet]. [cited 2021 Jan 30]. Available from: https://www.britannica.com/

3- Kollmann, M., Martins, W. P., & Raine-Fenning, N. (2014). Terms and thresholds for the ultrasound evaluation of the ovaries in women with hyperandrogenic anovulation. *Human reproduction update*, *20*(3), 463-464.

4- Loh, H. H., Yee, A., Loh, H. S., Kanagasundram, S., Francis, B., & Lim, L. L. (2020). Sexual dysfunction in polycystic ovary syndrome: a systematic review and meta-analysis. *Hormones*, *19*, 413-423.

5- Filippou, P., & Homburg, R. (2017). Is foetal hyperexposure to androgens a cause of PCOS?. *Human Reproduction Update*, *23*(4), 421-432.

6- Sam, S. (2015). Adiposity and metabolic dysfunction in polycystic ovary syndrome. *Hormone molecular biology and clinical investigation*, *21*(2), 107-116.

7- Laganà, A. S., Vitagliano, A., Noventa, M., Ambrosini, G., & D'Anna, R. (2018). Myo-inositol supplementation reduces the amount of gonadotropins and length of ovarian stimulation in women undergoing IVF: a systematic review and meta-analysis of randomized controlled trials. *Archives of Gynecology and Obstetrics*, *298*, 675-684.

8- Goodman, N. F., Cobin, R. H., Futterweit, W., Glueck, J. S., Legro, R. S., & Carmina, E. (2015). American Association of Clinical Endocrinologists, American College of Endocrinology, and androgen excess and PCOS society disease state clinical review: guide to the best practices in the evaluation and treatment of polycystic ovary syndrome-part 1. *Endocrine Practice*, *21*(11), 1291-1300.

9- Belfiore A, LeRoith PE. 2018. Principles of Endocrinology and Hormone Action. Cham: Springer; 2018. ISBN 9783319446752.

10- Campbell, M., & Jialal, I. (2019). Physiology, endocrine hormones.

11- Stamatiades, G. A., & Kaiser, U. B. (2018). Gonadotropin regulation by pulsatile GnRH: Signaling and gene expression. Molecular and Cellular Endocrinology, 463, 131141.

12- Plewes, M. R., Hou, X., Talbott, H. A., Zhang, P., Wood, J. R., Cupp, A. S., & Davis, J. S. (2020). Luteinizing hormone regulates the phosphorylation and localization of the mitochondrial effector dynamin-related protein-1 (DRP1) and steroidogenesis in the bovine corpus luteum. *The FASEB Journal*, *34*(4), 5299-5316.

13- Bishop, M. L., Fody, E. P., & Schoeff, L. E. (2010). Clinical chemistry: techniques, principles, correlations/[edited by] Michael L. Bishop, Edward P. Fody, Larry E. Schoeff.

14- Grinspon, R. P., & Urrutia, M. (2020). The importance of follicle-stimulating hormone in the prepubertal and pubertal testis. *Current Opinion in Endocrine and Metabolic Research*, *14*, 137-144.

15- Gharahdaghi, N., Phillips, B. E., Szewczyk, N. J., Smith, K., Wilkinson, D. J., & Atherton, P. J. (2021). Links between testosterone, oestrogen, and the growth hormone/insulin-like growth factor axis and resistance exercise muscle adaptations. *Frontiers in physiology*, *11*, 621226.

16- Kelly, D. M., & Jones, T. H. (2015). Testosterone and obesity. *Obesity Reviews*, *16*(7), 581-606.

17- Nagy, B., Szekeres-Barthó, J., Kovács, G. L., Sulyok, E., Farkas, B., Várnagy, Á., ... & Bódis, J. (2021). Key to life: physiological role and clinical implications of progesterone. *International journal of molecular sciences*, *22*(20), 11039.

18- González-Orozco, J. C., & Camacho-Arroyo, I. (2019). Progesterone actions during central nervous system development. *Frontiers in neuroscience*, *13*, 503.

19- Taraborrelli, S. (2015). Physiology, production and action of progesterone. *Acta obstetricia et gynecologica Scandinavica*, *94*, 8-16.

20- Rzeszowska, M., Leszcz, A., Putowski, L., Hałabiś, M., Tkaczuk-Włach, J., Kotarski, J., & Polak, G. (2016). Anti-Müllerian hormone: structure, properties and appliance. *Ginekologia polska*, *87*(9).

21- Visser, J. A., de Jong, F. H., Laven, J. S., & Themmen, A. P. (2006). Anti-Mullerian hormone: a new marker for ovarian function. *Reproduction*, *131*(1), 1-9.

22- Ali, M., & Mirza, L. (2021). Morbid obesity due to prolactinoma and significant weight loss after dopamine agonist treatment. *AACE Clinical Case Reports*, *7*(3), 204-206.

23- Porto-Neto, L. R., Bickhart, D. M., Landaeta-Hernandez, A. J., Utsunomiya, Y. T., Pagan, M., Jimenez, E., ... & Sonstegard, T. S. (2018). Convergent evolution of slick coat in cattle through truncation mutations in the prolactin receptor. *Frontiers in Genetics*, *9*, 57.

24- Deswal, R., Nanda, S., & Dang, A. S. (2019). Association of Luteinizing hormone and LH receptor gene polymorphism with susceptibility of Polycystic ovary syndrome. *Systems biology in reproductive medicine*, *65*(5), 400-408.

25- Atoum, M. F., Alajlouni, M. M., & Alzoughool, F. (2022). A case-control study of the luteinizing hormone level in luteinizing hormone receptor gene (rs2293275) polymorphism in polycystic ovarian syndrome females. *Public Health Genomics*, *25*(3-4), 89-97.

26- Barbieri, R. L., & Ehrmann, D. A. (2020). Treatment of polycystic ovary syndrome in adults. *Dostupno na: http://www. uptodate. com/contents/treatment-ofpolycystic-ovary-syndrome-in-adults*, 3-145.

27- Kumar, P., Sait, S. F., Sharma, A., & Kumar, M. (2011). Ovarian hyperstimulation syndrome. *Journal of human reproductive sciences*, *4*(2), 70-75.

28- Su, P., Chen, C., & Sun, Y. (2025). Physiopathology of polycystic ovary syndrome in endocrinology, metabolism and inflammation. *Journal of ovarian research*, *18*(1), 34.

29- Harrison, T. N., & Chang, R. J. (2022). Ovarian response to follicle-stimulating hormone in women with polycystic ovary syndrome is diminished compared to ovulatory controls. *Clinical endocrinology*, *97*(3), 310-318.

30- Xu, Y., Zhang, Z., Wang, R., Xue, S., Ying, Q., & Jin, L. (2024). Roles of estrogen and its receptors in polycystic ovary syndrome. *Frontiers in Cell and Developmental Biology*, *12*, 1395331.

31- Maybin, J. A., & Critchley, H. O. (2016). Medical management of heavy menstrual bleeding. *Women's Health*, *12*(1), 27-34.

32- Rasquin Leon, L. I., & Mayrin, J. V. (2017). Polycystic ovarian disease (Stein-Leventhal syndrome). *StatPearls. Treasure Island FL: StatPearls Publishing LLC*.

33- Joshi, A. (2024). PCOS stratification for precision diagnostics and treatment. *Frontiers in cell and developmental biology*, *12*, 1358755.

34- Marie, C., Pierre, A., Mayeur, A., Giton, F., Corre, R., Grynberg, M., ... & Chauvin, S. (2023). Dysfunction of human estrogen signaling as a novel molecular signature of polycystic ovary syndrome. *International Journal of Molecular Sciences*, *24*(23), 16689.

35- Jin, H., Yang, H., Zheng, J., Zhou, J., & Yu, R. (2023). Risk factors for low oocyte retrieval in patients with polycystic ovarian syndrome undergoing in vitro fertilization. *Reproductive Biology and Endocrinology*, *21*(1), 66..

36- van der Ham, K., Laven, J. S., Tay, C. T., Mousa, A., Teede, H., & Louwers, Y. V. (2024). Anti-Müllerian hormone as a diagnostic biomarker for Polycystic Ovary Syndrome (PCOS) and Polycystic Ovarian Morphology (PCOM): a systematic review and meta-analysis. *Fertility and sterility*..

37- Sumji, S., Bhat, A., Rashid, A., Bashir, R., Wani, I. A., Vasudevan, V., ... & Ganie, M. A. (2023). Efficacy of serum anti-mullerian hormone (AMH) levels for prediction of polycystic ovary syndrome (PCOS) and its association with clinical, biochemical and hormonal parameters. *Indian Journal of Clinical Biochemistry*, *38*(4), 457-465.

38- Vale-Fernandes, E., Pignatelli, D., & Monteiro, M. P. (2025). Should anti-Müllerian hormone be a diagnosis criterion for polycystic ovary syndrome? An in-depth review of pros and cons. *European Journal of Endocrinology*, *192*(4), R29-R43.

39- Saei Ghare Naz, M., Mousavi, M., Mahboobifard, F., Niknam, A., & Ramezani Tehrani, F. (2022). A meta-analysis of observational studies on prolactin levels in women with polycystic ovary syndrome. *Diagnostics*, *12*(12), 2924.

40- Hassan, M. F. (2020). The frequency of elevated prolactin level in polycystic ovary syndrome women (PCOS) and its' effect on pregnancy rate. *Global Journal of Public Health Medicine (GJPHM)*, *2*(1), 109-117.

41- Yang, H., Di, J., Pan, J., Yu, R., Teng, Y., Cai, Z., & Deng, X. (2020). The association between prolactin and metabolic parameters in PCOS women: a retrospective analysis. *Frontiers in endocrinology*, *11*, 263.

42- Mastnak, L., Herman, R., Ferjan, S., Janež, A., & Jensterle, M. (2023). Prolactin in polycystic ovary syndrome: metabolic effects and therapeutic prospects. *Life*, *13*(11), 2124.

43- Emanuel, R. H., Roberts, J., Docherty, P. D., Lunt, H., Campbell, R. E., & Möller, K. (2022). A review of the hormones involved in the endocrine dysfunctions of polycystic ovary syndrome and their interactions. *Frontiers in endocrinology*, *13*, 1017468.

IRAQI Academic Scientific Journals
Samarra Journal of Pure and Applied Science
SJPAS
مجلة سامراء للعلوم الصرفة والتطبيقية
www.sjpas.com
p ISSN: 2663-7405
e ISSN: 2789-6838

دراسة سريرية لتغيرات مستوى الهرمونات لدى النساء المصابات بمتلازمة تكيس المبايض في مدينة سامراء

ميسم ابراهيم مهدي [1]*، ريم ابراهيم مهدي[2]، ايات موفق مجيد[1]، زينب حسين علي[2]
1- قسم علوم الحياة، كلية التربية للعلوم الصرفة، جامعة سامراء، العراق
2- قسم الكيمياء، كلية التربية للعلوم الصرفة، جامعة سامراء، العراق

معلومات البحث:
تاريخ الاستلام: 2025/03/23
تاريخ التعديل: 2025/04/30
تاريخ القبـــول: 2025/05/02
تاريخ الـنشر: 2025/12/30
الكلمات المفتاحية:
تكيس المبايض،الهرمونات الجنسية،البرولاكتين،الهرمون المضاد لمولر.
معلومات المؤلف
الايميل:

الخلاصة:
هدفت الدراسة الحالية إلى تقييم تأثير متلازمة تكيس المبايض على مستويات مجموعة من الهرمونات التناسلية المختارة، والتي تشمل الهرمون المنشط للجسم الأصفر ، الهرمون المنشط للحوصلة ، الإستروجين، البروجسترون، الهرمون المضاد لمولر والبرولاكتين لدى نساء من سامراء، العراق. اجريت الدراسة من خلال اختيار 40 امرأة تم تقسيمهن إلى مجموعتين. مثّلت المجموعة الأولى النساء السليمات وتضمنت 15 امرأة، في حين مثّلت المجموعة الثانية مجموعة المريضات وتضمنت 25 امرأة، وذلك بعد تشخيصهن بمتلازمة تكيس المبايض من قبل طبيب مختص وإجراء الفحوصات اللازمة والسونار، وذلك خلال الفترة من أكتوبر 2024 إلى يناير 2025. تم قياس مستويات الهرمونات باستخدام اختبارات مصلية، وتم تحليل البيانات باستخدام اختبار (t-test) المستقل عبر برنامج SPSS الإصدار 25. أظهرت نتائج هذه الدراسة أن مجموعة النساء المصابات بمتلازمة تكيس المبايض لديهن مستويات أعلى بكثير من الهرمون اللوتيني، والإستروجين، والهرمون المضاد للمولر، والبرولاكتين، ومستويات أقل بكثير من الهرمون المنبه للجريب والبروجسترون مقارنة بالمجموعة الضابطة.