



Luteinizing Hormone Variability in Polycystic Ovary Syndrome: A Comparative Analysis

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ABSTRACT: An endocrine with metabolic condition known as Polycystic Ovarian Syndrome (PCOS) affects women who are of reproductive age, women with PCOS are a condition often associated with dysregulation of the hypothalamic-pituitary axis. A Cross-sectional study, the study included 88 patients who visited the outpatient clinic in total. These individuals were split up into two groups. All females are assessed the status of obesity by the body mass index (BMI), [BMI= weight (kg) / height (m)²], LH, FSH, Estradiol and Testosterone levels measured by ELISA technique. There was no discemible variation in BMI between groups A and B. (P \ge 0.456), The patients' mean FSH and LH values were 5.06 mIU/ml and 5.47 mIU/ml, compared with group B mean that equal to 6.05 mIU/ml, 18.91 mIU/ml respectively. Serum testosterone was slightly elevated in group B that was (41.19 \pm 5.16 ng/dL), and (39.53 \pm 4.98 ng/dL) in group A. Moreover, the level of estradiol (E2) in group A was 79.45 ng/dL while in group B the level was 72.65 ng/dL there are no significant difference between two groups. The study found conflicting evidence regarding the impact of high LH levels on oocyte quality, fertilization, and pregnancy outcomes in PCOS patients. According to the research, ovarian theca cells from PCOS patients may be more effective than normal theca cells at converting androgenic precursors to testosterone. While ovulatory dysfunction in PCOS is linked to reduced estradiol production, the study indicates that ovarian tissue may still contribute to circulating estradiol levels in affected women.

Keywords: Estradiol, Luteinizing hormone, Follicle stimulating hormone



1. INTRODUCTION

An endocrine and metabolic condition known as polycystic ovarian syndrome (PCOS) affects women who are of reproductive age [1]. is defined by elevated testosterone levels, hyperandrogenism, and chronic ovulatory failure. PCOS has been associated with luteinizing hormone, obesity, and insulin resistanc [2]. a clinical diagnosis that satisfies the Rotterdam Criteria and requires two of the following three symptoms: polycystic ovarian morphology (PCOM), hyperandrogenism, and/or oligo-anovulation [3].

A common complication of PCOS in women is hypothalamic-pituitary axis dysfunction [4]. The role of other endocrine variables, hyperactivity of the GnRH puke, and disturbance of the ovarian estrogen feedback mechanism are blamed for the increased release of LH and the typical drop in FSH [5]. Increased ovarian androgen production is caused by a changed LH-FSH ratio [6]. Regardless of dietary status, it has been demonstrated that decreased aromatase activity in PCOS leads to lower amounts of estradiol and increased testosterone in PCOS women [7].

The additional glandular conversion of androgens to estrogens, which occurs when these estrogens are not released from the ovary, is the cause of the excess estrogens in the blood. Due to the inability of the granulosa cells to form mature follicles, the polycystic ovary produces very little estrogen, which may be the cause of infertility in PCOS patients due to insufficient ovulation [8]. The aim of this study is to compare the high levels of LH in some women with PCOS to normal levels in other PCOS patients.

2. METHOD

From March 2023 to April 2024, a cross-sectional study was carried out in the Abu Ghraib General Hospital in Baghdad, Iraq, 88 patients were included. The practical side of the study was performed at the International Center for Development and Research (ICRD). The study included 88 patients who visited the outpatient clinic in total. These

subjects were divided into two groups, group A included 43 females have PCOS with normal level of Luteinizing hormone (LH) and the group B includes 45 females has PCOS with a high level of Luteinizing hormone (LH), the age was ranged between (16 - 36) years. All females are assessed the status of obesity by the body mass index (BMI), [BMI=weight (kg) / height (m)²], LH, FSH, Estradiol and Testosterone levels measured by ELISA technique as shown in figure 1.

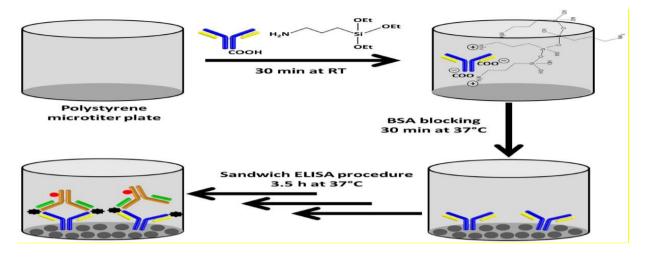


FIGURE 1. - ELISA sandwich technique

All subjects had a venous blood sample taken while seated using a disposable syringe (5 mL). Each individual had a vein puncture to get five milliliters of blood, which was then carefully injected into a simple tube. After centrifuging blood at 10000-x g for ten minutes, the serum was separated into three portions and kept at -20 $^{\circ}$ C until examination.

3. RESULT

3.1 Body Mass Index

There was no significant difference in BMI between group A and group B ($P \ge 0.456$), which may be due to the lifestyle of the population and e There was no discernible variation in BMI between groups A and B. at behaviors, therefore about 76.75% from group A and 51.1% from group B was obese among study subject, regarding group A considered obese while group B was overweight according to international standard as shown in table1.

3.2 Age group

The age difference between Group A and Group B was not statistically significant. ($P \ge 0.441$). The mean of age in group A was 24.25 years, while in group B 23.51 years.

3.3 Duration of Infertility

Between Group A and Group B, there was no discernible difference in the length of infertility. ($P \ge 0.489$), may be due to each group having PCOS and all women being obese, as illustrated in Table 1

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Group	BMI	Age (year)
	(kg/m2)	
Group A	31.66 ± 1.16	24.25 ± 0.78
Group B	29.82 ± 2.12	23.51 ± 0.57
P-value	0.456	0.441

Table 1. - Compare between Group A and Group B in different variables

3.4 Compare between the two groups in different hormones

The mean FSH and LH values in the patients in this study were 5.06 mIU/ml and 5.47 mIU/ml, compared with group B mean that equal to 6.05 mIU/ml, 18.91mIU/ml respectively.

Serum testosterone slightly elevated in group B that was $(41.19 \pm 5.16 \text{ ng/dl})$, and $(39.53 \pm 4.98 \text{ ng/dl})$ in group A. Moreover, the level of estradiol (E2) in group A was 79.45ng/dl whereas the level in group B was 72.65 ng/dl there are no significant difference between two groups, show Table 2.

Mean ± SE			
LH(mlU/ml)	FSH(mlU/ml)	Testo.(ng/dl)	E2 (pg/ml)
5.47 ± 0.49	5.06 ± 0.31	39.53 ± 4.98	79.45±8.17
18.91 ± 3.07	6.05 ± 0.34	41.19 ± 5.16	72.65±7.16
0.0001**	0.038*	0.81	0.53
	LH(mlU/ml) 5.47 ± 0.49 18.91± 3.07	LH(mlU/ml)FSH(mlU/ml) 5.47 ± 0.49 5.06 ± 0.31 18.91 ± 3.07 6.05 ± 0.34	LH(mlU/ml)FSH(mlU/ml)Testo.(ng/dl) 5.47 ± 0.49 5.06 ± 0.31 39.53 ± 4.98 18.91 ± 3.07 6.05 ± 0.34 41.19 ± 5.16

Table 2. - Compare between Group A and Group B in different variables

(* <0.05, **<0.001).

4. **DISCUSSION**

There was no discernible variation in BMI between groups A and B. ($P \ge 0.456$), may be due to life style of population and eat behaviors, therefore about 76.75% and 51.1% was obese among group A and B respectively, According Lowenstein et al [9] which found the BMI of the study have showed the group A was 31.66 kg/m2, while group B 28.82 kg/m2. Moreover, this study agreed with Akibyani et al, [10]. who obesity and overweight are common finding in PCOS, the There have been conflicting suggestions on the relationship between BMI and reproductive hormone levels.

This study agreement with Alnakash et al [11]. According to reports, PCOS is more common in younger women (under 35) than in older women. This may be because of a follicular physiological decrease that results in normalized ovarian function. with aging, an ultrasonographic look. Age can also affect PCOS's clinical presentation and metabolic symptoms, according to recent studies.

According to the study's findings, group A infertility lasted 35 months, while group B was 29.89 months. This study agreement with Brassard et al [12], with increasing age of the female partner and increasing duration of infertility (>3 years), conception is even less likely. Moreover, according to Femandez et al. [13], For women with PCOS, ovulation problems are usually the primary cause of infertility. In instances where ovulation is successful, hormonal imbalances may impede the appropriate growth of the uterine lining, hence impeding the mature egg's ability to implant. Menstrual cycles that are erratic and unexpected can also result from a hormonal imbalance, making pregnancy efforts more difficult.

The main cause of infertility in women with PCOS is typically irregular or unpredictable menstrual cycles. Even in cases where ovulation is successful, hormonal imbalances can hinder the development of the uterine lining, which is necessary for the implantation of the mature egg. Additionally, irregular or unpredictable menstrual cycles can further complicate the process of becoming pregnant. [14]. As well as reduced pregnancy and elevated miscarriage rates [15]. Furthermore, other research demonstrates that LH has no detrimental effects on the quality of oocytes and embryos, or on fertilization, implantation, or pregnancy [16]. Because the effects of lowering endogenous LH levels with GnRH agonists were likewise inconsistent, some researches have hypothesized that this strategy could lower the incidence of miscarriages [17]. Furthermore, this research aligns with the findings of Shi et al., who postulated that PCOS arises from the ovaries' fundamental incapacity to generate hormones in the appropriate ratios; the pituitary gland recognizes this abnormal ovarian function and responds by secreting atypical levels of both LH and FSH. These hormones are required for ovulation, the development of the ovary, and the release of an egg [18].

In addition, the result of this study was in agreement with the findings of previous studies like Meek et al [19], results suggest that polycystic ovarian disease patients' ovarian theca cells are superior to normal theca cells in their ability to convert androgenic precursors into testos terone. In addition, according to Als ibyani et al. and Iwas a et al. [10] [20]. which found the mean concentration of testos terone level was higher in patients with polycystic ovary syndrome, but no cutoff value was distinguished between the two groups by testos terone level only.

Ovulatory dysfunction in PCOS is linked to reduced estradiol production. However, research on granulosa cells from PCOS patients indicates that ovarian tissue may still contribute to circulating estradiol levels in affected women [21]. Since PCOS is associated with excessive androgen synthesis, it was interesting to find out if these androgens converted to 5- α -reduced metabolites that could inhibit aromatase activity. Local creation of E2 could partially explain PCOS's response to FSH. [22, 23].

5. CONCLUSION

There was no significant difference in BMI, age, or duration of infertility between the two groups of PCOS patients (those with normal LH levels and those with high LH levels). The study found conflicting evidence regarding the impact of high LH levels on oocyte quality, fertilization, and pregnancy outcomes in PCOS patients. According to the research, PCOS patients' ovarian theca cells may be more effective than normal theca cells at converting androgenic precursors to testosterone. While ovulatory dysfunction in PCOS is linked to reduced estradiol production, the study indicates that ovarian tissue may still contribute to circulating estradiol levels in affected women.

Overall, the study highlights the hormonal variability within PCOS patients, particularly in LH levels, while noting that other factors such as BMI, age, and infertility duration were not significantly different between the two groups.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest

REFERENCES

- [1] Y. Gu et al., "Life Modifications and PCOS: Old Story But New Tales," Front Endocrinol (Lausanne), vol. 13, p. 808898, 2022.
- [2] Z. Saadia, "Follicle stimulating hormone (LH: FSH) ratio in polycystic ovary syndrome (PCOS)-obese vs. nonobese women," Medical Archives, vol. 74, no. 4, pp. 289, 2020.
- [3] S. F. Witchel, A. C. Burghard, R. H. Tao, and S. E. Oberfield, "The diagnosis and treatment of PCOS in adolescents: an update," Current opinion in pediatrics, vol. 31, no. 4, pp. 562-569, 2019.
- [4] N. E. Baskind and A. H. Balen, "Hypothalamic-pituitary, ovarian and adrenal contributions to polycystic ovary syndrome," Best Practice & Research Clinical Obstetrics & Gynaecology, vol. 37, pp. 80-97, 2016.
- [5] Q. Xia et al., "Elevated baseline LH/FSH ratio is associated with poor ovulatory response but better clinical pregnancy and live birth in Chinese women with PCOS after ovulation induction," Heliyon, vol. 9, no. 1, p. e13024, 2023.
- [6] M. S. Morshed et al., "Luteinizing hormone to follicle-stimulating hormone ratio significantly correlates with androgen level and manifestations are more frequent with hyperandrogenemia in women with polycystic ovary syndrome," Journal of Endocrinology and Metabolism, vol. 11, no. 1, pp. 14-21, 2021.
- [7] G. Franik, M. Maksym, A. J. Owczarek, J. Chudek, P. Madej, and M. Olszanecka-Glinianowicz, "Estradiol/testosterone and estradiol/androstenedione indexes and nutritional status in PCOS women–A pilot study," European Journal of Obstetrics & Gynecology and Reproductive Biology, vol. 242, pp. 166-169, 2019.
- [8] M. Khattak, N. Sultana, A. F. Khattak, R. Usman, and A. Khattak, "Comparison of Estradiol levels in Polycystic Ovary Syndrome and non-Polycystic Ovary Syndrome Infertile Patients," Advances in Basic Medical Sciences, vol. 6, no. 1, pp. 20-23, 2022.
- [9] E. J. Lowenstein, "Diagnosis and management of the dermatologic manifestations of the polycystic ovary syndrome," Dermatologic therapy, vol. 19, no. 4, pp. 210-223, 2006.
- [10] N. A. Alsibyani et al., "Clinical Presentation of Polycystic Ovary Syndrome among Saudi Arabian Women-Jeddah, Saudi Arabia," The Egyptian Journal of Hospital Medicine, vol. 31, no. 71, pp. 1-5, 2017.
- [11] A. H. Alnakash and N. K. Al-Tae'e, "Polycystic ovarian syndrome: the correlation between the LH/FSH ratio and disease manifestations," Middle East Fertility Society Journal, vol. 12, no. 1, p. 35, 2007.
- [12] M. Brassard, Y. AinMelk, and J-P. Baillargeon, "Basic infertility including polycystic ovary syndrome," Medical Clinics of North America, vol. 92, no. 5, pp. 1163-1192, 2008.
- [13] R. C. Fernandez et al., "Sleep disturbances in women with polycystic ovary syndrome: prevalence, pathophysiology, impact and management strategies," Nature and science of sleep, vol. 10, p. 45, 2018.
- [14] A. H. Balen, S-L. Tan, J. MacDougall, and H. S. Jacobs, "Miscarriage rates following in-vitro fertilization are increased in women with polycystic ovaries and reduced by pituitary desensitization with buserelin," Human Reproduction, vol. 8, no. 6, pp. 959-964, 1993.
- [15] R. Homburg, "Pregnancy complications in PCOS," Best Practice & Research Clinical Endocrinology & Metabolism, vol. 20, no. 2, pp. 281-292, 2006.
- [16] C. Mendoza et al., "Follicular fluid markers of oocyte developmental potential," Human Reproduction, vol. 17, no. 4, pp. 1017-1022, 2002.

- [17] T. Haahr, M. Roque, S. C. Esteves, and P. Humaidan, "GnRH agonist trigger and LH activity luteal phase support versus hCG trigger and conventional luteal phase support in fresh embryo transfer IVF/ICSI cycles—a systematic PRISMA review and meta-analysis," Frontiers in endocrinology, vol. 8, p. 116, 2017.
- [18] X. Shi, L. Zhang, S. Fu, and N. Li, "Co-involvement of psychological and neurological abnormalities in infertility with polycystic ovarian syndrome," Archives of gynecology and obstetrics, vol. 284, no. 3, pp. 773-778, 2011.
- [19] S. Shabbir et al., "The interplay between androgens and the immune response in polycystic ovary syndrome," Journal of Translational Medicine, vol. 21, no. 1, p. 259, Apr. 2023.
- [20] T. Iwasa et al., "Diagnostic performance of serum total testosterone for Japanese patients with polycystic ovary syndrome," Endocrine journal, p. 0702050030, 2007.
- [21] K. J. Hamilton, S. C. Hewitt, Y. Arao, and K. S. Korach, "Estrogen hormone biology," in Current topics in developmental biology, vol. 125, Elsevier, 2017, pp. 109-146.
- [22] D. A. Ehrmann, "Polycystic ovary syndrome," New England Journal of Medicine, vol. 352, no. 12, pp. 1223-1236, 2005.
- [23] R. L. Barbieri and W. F. Crowley Jr, "Steroid hormone metabolism in polycystic ovary syndrome," *UpToDate*, P. J. Snyder and W. F. Crowley.