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Hormonal Imbalance of Estrogen and Progesterone, Its Association with PCOS, and Its Impact on Pregnancy Rates in Women from Thi-Qar Governorate

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***Abstract*— Polycystic Ovary Syndrome (PCOS) is a common endocrine disorder-affecting women of reproductive age, characterized by hormonal imbalances, irregular ovulation, and metabolic disturbances. The variability in estrogen and progesterone levels plays a crucial role in reproductive health. The aim of this study is to evaluate the levels of Estradiol-E2, progesterone (P4), and their effect on pregnancy rates in women with PCOS. It also covers the effect of follicle-stimulating hormone (FSH), luteinizing hormone (LH), testosterone, thyroid-stimulating hormone (TSH) and anti-Müllerian hormone (AMH). Additionally, metabolic markers such as fasting glucose, fasting insulin, body mass index (BMI), and insulin resistance (HOMA-IR). This study was conducted on 120 women aged 18-45 years, categorized into two age groups (18-30 and 31-45 years) to ensure balanced representation (60 with PCOS and 60 healthy controls). Serum hormone levels were measured using ELFA techniques, and metabolic parameters were evaluated using standard biochemical assays. PCOS patients exhibited significantly higher levels of (E2), LH, AMH, and testosterone, while progesterone and FSH were significantly lower compared to the control group ($P < 0.05$). HOMA-IR levels were significantly higher in PCOS patients, indicating an increased prevalence of insulin resistance. Pearson correlation analysis revealed a strong positive correlation between HOMA-IR and Estradiol-E2 ($r = 0.558$, ** $P < 0.05$) and a strong negative correlation with progesterone ($r = -0.654$, ** $P < 0.05$). The hormonal and metabolic dysregulations in PCOS negatively affect pregnancy rates, with Estradiol-E2, progesterone, and insulin resistance playing key roles. Understanding these associations can enhance treatment strategies to improve fertility outcomes in PCOS.**

***Keywords*— Estradiol-E2, Progesterone, PCOS, HOMA-IR, Pregnancy Rates.**

I. INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common endocrine disorder affecting 6–12% of women of reproductive age, who present with hyperandrogenism, ovulatory dysfunction, and polycystic ovaries [1- 3]. PCOS affects women of all ages. It primarily manifests itself as menstruation problems, hirsutism, and acne, together with infertility, and pregnancy issues [4]. Ovulatory dysfunction, one of the criteria for PCOS diagnosis [5]. However, several experimental and clinical findings underline that the fertility in women with PCOS may also be affected by abnormalities in endometrial and oocyte [6]. Impact of PCOS on pregnancy rates: The multiple endocrine-metabolic disturbances in PCOS have potential detrimental effects on folliculogenesis and oocyte maturation [7, 8]. Substantial differences have been documented between oocytes from PCOS women and oocytes from healthy women. MII oocytes from PCOS patients had an altered gene expression profile [9]. Role of estrogen and progesterone in reproductive health: Estrogen is a key hormone that promotes the growth and preparation of the endometrium for embryo implantation, regulating physiological and pathological processes in the reproductive. Therefore, it is also involved with scores of diseases, for example, infertility, PCOS. With PCOS have elevated estrogen levels; these levels may be unstable, leading to abnormal endometrial growth and an increased risk of endometrial hyperplasia [10]. Progesterone is a hormone that is essential for implantation and maintenance of early human pregnancy. The follicular phase of the menstrual cycle is dominated by estrogen, while the luteal phase of the menstrual cycle is dominated by progesterone [11]. The secretion of progesterone transforms the proliferative endometrium, activated by estrogen, into a secretory endometrium that is receptive to the blastocyst. Prior to ovulation, granulosa cells in the follicle biosynthesize and secrete estrogen. After the follicle ruptures and releases the egg, these granulosa cells mature and form the CL, which is responsible for secreting progesterone and estrogen during the final part of the cycle [12]. Impact of hormonal imbalances on pregnancy: (PCOS) can cause menstrual irregularities, hyperandrogenism, impaired fertility, polycystic ovaries, and metabolic irregularities like elevated levels of luteinizing hormone (LH), testosterone, and insulin, as well as reduced levels of follicle-stimulating hormone (FSH) [13]. PCOS is characterized by a high level of androgen and fits the hyperandrogenism criteria. According to the NIH consensus criteria, around 80% of

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women with polycystic ovaries have elevated testosterone levels[14]. TSH, thyroid abnormalities are particularly frequent in women and can cause menstruation disruption, infertility, and metabolic disorders [15, 16,17]. AMH is involved in basic follicular development and is most likely responsible for follicle selection. According to research, blood AMH levels may provide significant information in sufferers with altered ovarian function, such as anovulation [18]. Miscarriage rates in women with pcos. Noteworthy PCOS seems to have a substantial negative effect on fertility. Due to disturbances in follicular maturation, follicle growth often stops at a follicle size of 4–8 mm, which prevents the development of a dominant follicle and, therefore, ovulation. In addition, once a pregnancy is achieved, spontaneous miscarriages can also be observed more frequently in PCOS patients [19]. The aim of this study is to evaluate estradiol (E2) and progesterone levels in women with and without PCOS and Their impact on pregnancy rates.

II. METHODOLOGY

A. Design of the study

This cross-sectional analytical study was conducted in Thi Qar Governorate from March 2023 to January 2024. The study aimed at assessing changes in Estradiol - E2 and progesterone levels among women diagnosed with polycystic ovary syndrome (PCOS), and examining their impact on pregnancy rates.

B. Study Samples

Sample Size**:** The study included 120 women of reproductive age (18-45 years), selected from Bint Al-Huda Teaching Hospital, Al-Haboubi Teaching Hospital, and Sumer Health Center, and divided into two groups:
Study Group: 60 women diagnosed with PCOS. Control Group**:** 60 women with regular menstrual cycles and no history of infertility or hormonal disorders.

C. Inclusion Criteria

Women aged 18-45 years.
Diagnosis of PCOS based on Rotterdam criteria at least two of the following criteria:
Oligo-ovulation or anovulation.
Clinical or biochemical signs of hyperandrogenism.
Polycystic ovaries on ultrasound (>12 follicles in one ovary) Based on the Adams criteria.
No other endocrine disorders, such as thyroid dysfunction or hyperprolactinemia.

D. Age Group Classification

All participants were within the reproductive age range of 18 to 45 years. For organizational purposes and to ensure balanced representation across age brackets, the participants were grouped into two main age categories:
- Group 1: 18-30 years (younger reproductive age)
- Group 2: 31-45 years (advanced reproductive age)

This categorization was used to ensure an even distribution of age within the study sample.

E. Exclusion Criteria

Women with other conditions affecting fertility (Type 1 or Type 2 diabetes, thyroid disorders, Cushing's syndrome).
Women who have used hormonal medications or contraceptives within the past three months.
Pregnant women or those with chronic conditions affecting hormonal balance.

F. Data Collection Methods

Clinical Interviews: Personal interviews were conducted in medical centers to document medical history, menstrual cycle patterns, ovulation disorders, and clinical symptoms of PCOS.

G. Physical Examinations

Body Mass Index (BMI) measurement, Hyperandrogenism related symptoms (acne, hair thinning, excessive hair growth - Hirsutism).

H. Laboratory Analysis Methods

Approximately 5 ml of venous blood was collected from each participant using gel separator tubes. The samples were centrifuged at 1,500 ×g for 10 minutes to obtain serum. The samples were then stored in a deep freezer in the central blood bank at -20°C to assess hormone levels. Hormone levels were assessed using ELFA (Enzyme-Linked Fluorescence Assay) ,and CLIA was used to measure fasting insulin. Spectrophotometry was used to measure fasting blood sugar. The values are used to calculate the insulin resistance index (HOMA-IR) using the equation: HOMA-IR=$\frac{\text{Fasting Insulin}(\mu U/mL)\times\text{Fasting Glucose}(mmol/L)}{22.5}$.

I. Ultrasound Examination (Transvaginal Ultrasound)

Transvaginal ultrasound was performed to evaluate ovarian morphology and determine polycystic ovary presence based on Adam's criteria, where an ovary was classified as polycystic if it contained more than 12 small follicles (2-9 mm) or had a volume >10 cm^3.

J. Data Analysis

Data were analyzed using SPSS version 26, employing the following statistical methods: descriptive statistics to calculate the mean hormone levels and standard deviations in both groups, and Pearson correlation analysis to explore the relationship between insulin resistance and reproductive hormone levels, ANOVA analysis to compare the differences between women with PCOS and the control group. Results were presented as mean ± standard deviation (SD), with statistical significance set at $P < 0.05$.

III. RESULTS

The table below compares women with polycystic ovary syndrome (PCOS) and women without PCOS (control group) in terms of demographic characteristics, metabolic Parameters, reproductive hormones, and all P-values were less than 0.05. Women with PCOS have weight gain, insulin resistance, and elevated androgen levels compared to the control group. Low progesterone and FSH levels and elevated Estradiol-E2, and LH reflect ovulation disorders associated with PCOS. High HOMA-IR indicates impaired glucose metabolism, which increases the risk of

complications such as type 2 diabetesdicating statistically significant differences between the two groups as in Table 1.

Table 1: Comparison of variables between the PCOS and control groups.

Variable	Mean ± SD N= 60 (PCOS)	Mean ± SD N= 60 (Control)	P-value
Age (in years)	25.58 ± 4.14	28.06 ± 3.10	$p < 0.05$
BMI (kg/m²)	28.39 ± 2.76	23.95 ± 2.28	$p < 0.05$
Fasting Glucose (mg/dL)	95.02 ± 8.87	84.63 ± 5.34	$p < 0.05$
Fasting Insulin (µU/mL)	14.78 ± 5.14	8.22 ± 2.10	$p < 0.05$
HOMA-IR	3.42 ± 1.17	1.73 ± 0.46	$p < 0.05$
Estradiol - E2(Pg/ml)	77.65 ± 13.60	49.96 ± 9.43	$p < 0.05$
Progesterone (ng/ml)	4.14 ± 1.42	10.11 ± 2.07	$p < 0.05$
TSH (MIU/ml)	2.75 ± 0.80	1.88 ± 0.51	$p < 0.05$
FSH (MIU/ml)	5.10 ± 0.90	7.58 ± 1.34	$p < 0.05$
LH (MIU/ml)	11.88 ± 3.05	6.22 ± 1.44	$p < 0.05$
Testosterone (ng/ml)	0.79 ± 0.20	0.40 ± 0.11	$p < 0.05$
AMH (ng/ml)	6.87 ± 2.25	3.11 ± 1.16	$p < 0.05$

SD: Standard deviation, BMI: body mass index , HOMA-IR : homeostatic model assessment of insulin resistance **,** follicle-stimulating hormone (FSH), luteinizing hormone (LH), thyroid-stimulating hormone (TSH), and anti-Müllerian hormone (AMH)

Table 2: Normal estradiol-E2 and progesterone levels compared in PCOS during the menstrual cycle.

Menstrual Phase	Normal Estradiol (E2) *(pg/mL)*	PCOS – E2 Range	Normal Progesterone (P4) *(ng/mL)*	PCOS – P4 Range	Clinical Interpretation in PCOS
Follicular Phase	20 – 150	60 – 120 ↑	< 1.5	~1 – 3 ↑	E2 may be elevated and unstable → endometrial overgrowth; P4 remains low due to anovulation
Ovulatory Peak	150 – 400	No significant peak	1.5 – 3	Often < 2 ↓	No LH surge → E2 fails to peak → ovulation does not occur
Luteal Phase	100 – 250	70 – 150 ↓ or flat	10 – 20	< 3 ↓	Low P4 confirms anovulation or luteal phase insufficiency

Table2 presents the normal hormonal levels for healthy women, which significantly different women with Polycystic Ovary Syndrome (PCOS)

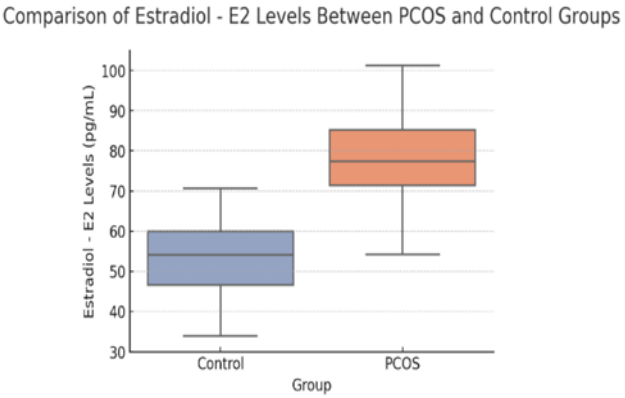

Fig. 1: Comparison of Estradiol-E2, levels between pcos and control groups.

Figure1 shows that women with PCOS had higher levels of Estradiol-E2 than the control group, which may contribute to ovulation disorders and irregular menstrual cycles. This increase may have an impact on fertility and the chances of pregnancy.

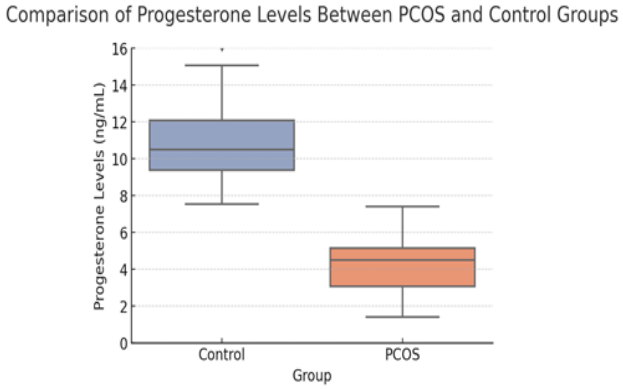

Fig. 2: Comparison of progesterone levels between the pcos and control groups.

Figure2 shows that women with PCOS had lower levels of progesterone than the control group, which may have a negative impact on pregnancy and fertility, given progesterone's role in preparing the uterine lining for pregnancy.

Table3: Correlations HOMA-IR with multiple reproductive hormones in PCOS.

Variable	F-statistic	P-value
BMI (Body Mass Index)	1524.34	2.77×10^{-248}
Fasting Glucose (mg/dL)	898.65	2.32×10^{-163}
Fasting Insulin (µU/mL)	1341.85	3.48×10^{-225}
HOMA-IR (Insulin Resistance Index)	1687.75	5.71×10^{-268}
Estradiol - E2	2752.58	0.0
Progesterone	5531.87	0.0
TSH	729.17	3.60×10^{-137}
FSH	2378.48	0.00
LH	3127.41	0.0
AMH (Anti-Müllerian Hormone)	2316.55	0.0

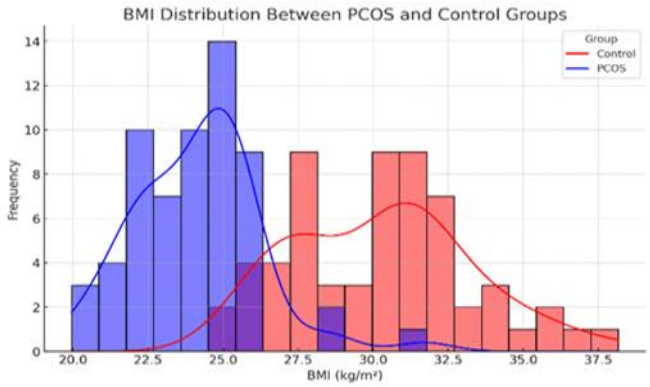

Fig. 3: BMI distribution between PCOS and control groups.

PCOS group (red histogram & red density curve) has a higher BMI distribution than the Control group (blue histogram & blue density curve).
PCOS patients tend to have higher BMI values, with some cases exceeding 35 kg/m², whereas most of the control group remains below 30 kg/m².

Table 4: ANOVA analysis comparing metabolic and hormonal parameters between PCOS patients and the control group.

Hormone	Correlation	P-value
Estradiol - E2 (Pg/ml)	0.558	**p < 0.05
Progesterone (ng/ml)	-0.654	**p < 0.05
TSH (MIU/ml)	0.419	**p < 0.05
FSH (MIU/ml)	-0.478	**p < 0.05
LH (MIU/ml)	0.545	**p < 0.05
Testosterone (ng/ml)	0.567	**p < 0.05
AMH (ng/ml)	0.554	**p < 0.05

This table presents the ANOVA analysis results comparing metabolic and hormonal parameters between PCOS patients and the control group. The F-statistic indicates the magnitude of the difference between the two groups, while the p-value determines statistical significance. All variables show significant differences ($p < 0.05$), confirming that metabolic and hormonal alterations in PCOS patients are distinct from those in healthy individuals. The most notable differences are observed in progesterone ($F = 5531.87$), estradiol ($F = 2752.58$), and insulin resistance ($F = 1687.75$), emphasizing their role in ovarian dysfunction and metabolic disturbances associated with PCOS. The following section discusses how previous studies explain these differences, as well as their clinical history and potential implications for the chances of pregnancy in women with PCOS.

IV. Discussion

The results of this study are consistent with previous research suggesting that hormonal imbalance in PCOS contributes to metabolic dysfunction and decreased fertility. A study by G. V. Callard *et al.* found that estrogen is a key hormone that promotes the growth of the endometrium and prepares it for implantation of the embryo. If estrogen levels are elevated in women with PCOS, these levels may be unstable, leading to abnormal growth of the endometrium and an increased risk of endometrial hyperplasia, which is consistent with our findings that elevated estrogen levels in PCOS patients [10]. Our study also showed that the decrease in progesterone level was more obvious, indicating more severe ovulation disorder. This is consistent with a study was done X. Bai *et al.* The secretion of progesterone and estrogen in PCOS patients is different from that in the normal population. Due to persistent ovulatory disorders and poor corpus luteum development in PCOS patients, progesterone levels are lower than those in non-PCOS patients, as shown in Table 1 [20]. The LH-FSH ratio is unbalanced in PCOS women. Compared with FSH, LH levels are too high, resulting in overstimulation of androgen (testosterone) secreting cells in the ovaries, disrupting the growth of follicles and preventing normal ovulation. This is consistent with the findings of Balen *et al.* The normal reproductive axis of women with PCOS is disrupted, so LH levels are increased and FSH levels are decreased, resulting in an inverted LH/FSH ratio [21]. In addition, the results showed that PCOS patients had increased testosterone levels compared with the control group ($P < 0.05$). Increased androgens,
combined with insulin resistance, lead to follicular stagnation and low endometrial receptivity, resulting in reduced fertility, which is consistent with the study of Z. Tang, *et al.* Women with PCOS have increased production of LH and free testosterone [22]. Studies by P. Pigny *et al.* and J.S.E. Lavine *et al.* have shown that higher anti-mullerian hormone levels were observed in women with PCOS. Serum AMH levels were significantly higher in

women with anovulation (World Health Organization or WHO2, category 2) compared with premenopausal women with normal ovulation, especially in women with polycystic ovarian morphology. These studies support our findings that women with PCOS have higher AMH levels than healthy women [23, 24].

How does insulin resistance affect hormonal imbalance?

The interaction between hormonal imbalance and insulin resistance in PCOS is well known. A study by C. A. Amisi, J. J. Kim *et al*. and S. H. Hong *et al.* showed that insulin resistance is a prominent feature of PCOS, affecting approximately 80% of cases. However, the prevalence of insulin resistance in women with PCOS varies widely due to different insulin resistance measurements and cutoff points. Insulin resistance in PCOS is primarily caused by androgen excess, which alters insulin activity in adipocytes and skeletal muscle. This pattern was also observed in our PCOS cohort and plays a central role in hormonal imbalance and ovulatory dysfunction in women with this syndrome, as shown in Table 3. This study found significant correlations between the HOMA-IR scale and several reproductive hormones, indicating their influence on hormonal balance, particularly in PCOS.

HOMA-IR was positively correlated with:

Estradiol-E2 (r = 0.558) → Increased insulin resistance leads to increased estrogen levels and contributes to hormonal imbalance in women with PCOS.

LH (r = 0.545) → Indicates the effect of insulin resistance on ovulation irregularity, as elevated LH stimulates excessive androgen production, which inhibits follicular growth.

Testosterone (r = 0.567) → Insulin resistance increases androgen (testosterone) levels, exacerbating symptoms such as acne and excessive hair growth (hirsutism).

AMH (r = 0.554) → Increased ovarian reserve in PCOS patients, as increased AMH levels reflect a greater number of immature follicles due to hormonal dysregulation.

TSH (r = 0.419) → the potential impact of insulin resistance on thyroid function, which may explain the increased risk of thyroid dysfunction in some PCOS patients.

HOMA-IR is negatively correlated with:

Progesterone (r = -0.654) → Indicates the impact of insulin resistance on ovulatory dysfunction, as abnormal progesterone levels contribute to menstrual irregularities and decreased pregnancy success rates.

FSH (r = -0.478) → Low FSH levels impair follicular maturation, leading to anovulation and menstrual irregularities in women with PCOS [25, 26, 27]. According to a study by T. M. Barber *et al* and S. T. Yang *et al.* the majority of women with PCOS have a high body mass index (BMI), and are often classified as overweight (BMI ≥ 25 kg/ m^2) or obese (BMI ≥ 30 kg/ m^2), with a prevalence of 38–88%. A high prepregnancy BMI is associated with adverse pregnancy outcomes in women with PCOS, regardless of the fertility treatment used. These studies are consistent with the findings of our study, in which women with PCOS tend to be overweight and have a higher BMI compared with healthy women. Being overweight increases insulin resistance, which leads to higher levels of insulin in the blood, which stimulates the ovaries to produce more androgens. High androgens cause irregular or absent menstrual cycles, poor ovulation, or infertility as listed in Figure 3 [28, 29].

V. RECOMMENDATIONS

Therapeutic interventions should focus on improving hormonal balance and insulin sensitivity through:

Weight management to mitigate the impact of obesity on insulin resistance.

Hormonal treatments to stimulate ovulation and enhance progesterone levels.

The use of insulin-sensitizing agents such as metformin to improve pregnancy outcomes in PCOS patients.

CONCLUSIONS

This study confirms that the hormonal and metabolic dysregulations in PCOS negatively affect pregnancy rates, with Estradiol-E2, progesterone, and insulin resistance playing key roles. Understanding these associations can enhance treatment strategies to improve fertility outcomes in pcos.

CONFLICT OF INTEREST

The author declares that there is no conflict of interest.

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