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Research Article

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Investigating the Role of Hormonal Imbalances in Polycystic Ovary Syndrome (PCOS) and Fertility Outcomes

Khondokar Seheli Nasrin Lina*[1]

1 Department of Gynae and Obstetrics, Rajshahi Medical College, Rajshahi, Bangladesh

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ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) is a multifactorial endocrine disorder causing ovulatory dysfunction and infertility, strongly linked to hormonal imbalance and metabolic disturbances in reproductive-aged women. ***Objective:*** This study aims to investigate hormonal imbalances in PCOS patients and analyze their impact on ovulatory function, oocyte quality, and fertility outcomes using clinical, biochemical, and statistical parameters. ***Methods:*** A cross-sectional observational study was conducted at the Department of Gynae and Obstetrics, Rajshahi Medical College, Bangladesh, from January to December 2023. One hundred PCOS-diagnosed patients (Rotterdam criteria) aged 18–35 years were enrolled. Hormonal assays (LH, FSH, LH/FSH ratio, total testosterone, fasting insulin) and metabolic profiles (BMI, HOMA-IR) were measured. Ovarian morphology was assessed by transvaginal ultrasound. Fertility outcomes, including ovulation rate, oocyte quality, and conception rates, were recorded. Data were analyzed using SPSS v.26 with t-tests and chi-square; significance was set at $p<0.05$. ***Results:*** Mean LH/FSH ratio was 2.8 ± 0.9, with elevated LH levels in 68% of patients. Hyperandrogenism was observed in 72%, while insulin resistance (HOMA-IR>2.5) was detected in 64%. Anovulation occurred in 61%, with 39% achieving ovulation. Oocyte maturation rates were 58% in PCOS patients versus reference 82% in controls ($p=0.004$). Fertility outcomes showed 32% conception within one year. Higher BMI (>27 kg/m²) was significantly associated with reduced ovulation ($p=0.03$) and lower conception ($p=0.02$). Standard deviation analysis demonstrated greater variability in LH (±7.2 IU/L) compared to FSH (±3.4 IU/L). Correlation revealed strong negative association between insulin resistance and oocyte quality ($r=-0.63$, $p=0.001$). ***Conclusion:*** Hormonal imbalance, particularly elevated LH/FSH ratio and insulin resistance, significantly impairs ovulation and fertility in PCOS, highlighting the necessity of integrated endocrine-metabolic management.

Keywords: PCOS, Hormonal Imbalance, Infertility, Insulin Resistance, Ovulation.

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INTRODUCTION

Polycystic Ovary Syndrome (PCOS) represents one of the most prevalent endocrine disorders among women of reproductive age, affecting an estimated 6–20% globally depending on diagnostic criteria applied.[1] It is a heterogeneous condition with multifactorial etiology, characterized by chronic anovulation, hyperandrogenism, and polycystic ovarian morphology.[2] Clinically, PCOS manifests as menstrual irregularities, subfertility, metabolic disturbances, and dermatological complications such as acne and hirsutism.[3] Despite its high prevalence, the pathophysiological mechanisms underpinning PCOS remain incompletely elucidated, and the interplay between hormonal imbalances and fertility outcomes is a critical area of ongoing investigation. The hormonal dysregulation observed in PCOS is complex, involving aberrant interactions between gonadotropins, sex steroids, insulin, and adipokines. Dysregulation of the hypothalamic–pituitary–ovarian (HPO) axis plays a central role, particularly through an altered secretion pattern of gonadotropin-releasing hormone (GnRH) and consequent luteinizing hormone (LH) hypersecretion.[4] Elevated LH levels promote excessive thecal androgen production, while inadequate follicle-stimulating hormone (FSH) activity impairs follicular development and

*Corresponding Authors:
Email: nasrinseheli@gmail.com

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ovulation.[5] Moreover, hyperandrogenism exacerbates anovulation and induces further metabolic and endocrine disruptions, creating a vicious cycle that perpetuates infertility.[6] Insulin resistance, an established hallmark of PCOS, further complicates the endocrine profile. Hyperinsulinemia enhances ovarian androgen production by amplifying LH-stimulated steroidogenesis and simultaneously suppresses hepatic production of sex hormone-binding globulin (SHBG), increasing circulating free androgens.[7] The co-existence of obesity in many PCOS patients amplifies this effect, linking metabolic dysfunction with reproductive abnormalities.[8] Such findings suggest that fertility impairment in PCOS is not only the consequence of ovarian dysfunction but also of systemic endocrine dysregulation with contributions from metabolic pathways.[9]

Reproductive dysfunction in PCOS is chiefly characterized by anovulation and impaired folliculogenesis. Ultrasonographic evidence reveals a large pool of small antral follicles in affected ovaries, yet the progression to dominant follicle selection and ovulation is disrupted.[10] The imbalance between LH and FSH disrupts granulosa cell proliferation and estradiol synthesis, ultimately impeding the acquisition of oocyte competence.[11] These disturbances are reflected in suboptimal fertility outcomes, both in natural conception and assisted reproductive technologies (ART).[12] Notably, oocyte quality, endometrial receptivity, and early embryonic development appear compromised in PCOS, leading to lower implantation and live birth rates.[13] Hormonal imbalances also exert a profound influence on the endometrium. Progesterone resistance, a frequently reported phenomenon in PCOS, impairs endometrial receptivity and decidualization.[14] Elevated androgens and insulin may modulate gene expression patterns within the endometrium, disrupting the delicate molecular cross-talk required for successful implantation.[15] Furthermore, aberrant estradiol signaling can lead to endometrial hyperplasia and increase the risk of endometrial carcinoma, further complicating reproductive outcomes.[16]

The molecular underpinnings of PCOS-associated hormonal imbalances are currently under active exploration. Polymorphisms in genes encoding steroidogenic enzymes, insulin receptor substrates, and gonadotropin receptors have been implicated in susceptibility to PCOS.[17] Increased expression of cytochrome P450c17α (CYP17A1), which catalyzes androgen biosynthesis, has been observed in thecal cells of PCOS patients.[18] Moreover, dysregulated intraovarian signaling pathways, including phosphatidylinositol 3-kinase (PI3K)/Akt and mitogen-activated protein kinase (MAPK), contribute to abnormal follicular dynamics [19]. The interaction of these molecular mechanisms with systemic factors such as obesity, inflammation, and oxidative stress underscores the multifactorial complexity of PCOS.[20] In addition to gonadal abnormalities, neuroendocrine dysregulation is increasingly recognized as pivotal in PCOS pathophysiology. Women with PCOS often exhibit increased GnRH pulse frequency, favoring LH over FSH secretion.[21] This abnormal pulsatility may arise from altered hypothalamic sensitivity to steroid feedback or disruptions in the activity of kisspeptin neurons, which are critical regulators of GnRH secretion.[22] Such evidence reinforces the need to consider PCOS as a systemic neuroendocrine disorder rather than a purely ovarian pathology.

Fertility outcomes in women with PCOS are deeply influenced by the extent of hormonal imbalance and metabolic dysfunction. While some individuals with mild phenotypes may achieve spontaneous conception, others require ovulation induction, ART, or adjunctive metabolic therapies.[23] Clinical interventions often target the hormonal milieu: clomiphene citrate and letrozole are employed to restore ovulation, while insulin sensitizers such as metformin address the metabolic derangements that exacerbate reproductive dysfunction.[24] ART outcomes in PCOS patients remain heterogeneous, with higher risks of ovarian hyperstimulation syndrome (OHSS) and variable implantation success.[25] Importantly, emerging data suggest that correction of metabolic abnormalities, such as weight reduction and improvement of insulin sensitivity, positively modulates hormonal balance and fertility outcomes.[26] Lifestyle modifications and pharmacological interventions therefore represent essential adjuncts to conventional fertility treatments in PCOS. Nonetheless, the precise relationships between the degree of hormonal imbalance, metabolic dysfunction, and reproductive potential remain incompletely characterized and require rigorous scientific evaluation.

MATERIAL AND METHODS

This investigation was designed as a cross-sectional observational study and was conducted in the Department of Gynaecology and Obstetrics, Rajshahi Medical College, Rajshahi, Bangladesh. The study period extended from January 2023 to December 2023. One hundred female patients between 18 and 35 years of age who were diagnosed with Polycystic Ovary Syndrome (PCOS) according to the revised Rotterdam criteria (2003) were included. Patients with confounding endocrine disorders such as thyroid dysfunction, hyperprolactinemia, or congenital adrenal hyperplasia were excluded to reduce bias. The study emphasized evaluating hormonal imbalances and their relationship with fertility outcomes, specifically ovulation, oocyte quality, and conception rates. Each participant underwent clinical examination, biochemical testing, and ultrasonographic assessment to ensure accurate diagnosis. A standardized protocol was followed to obtain baseline demographic data, anthropometric indices, reproductive history, and relevant laboratory markers. The design allowed systematic observation and statistical interpretation of endocrine-metabolic disturbances in relation to reproductive parameters. Data were collected using structured case record forms that captured demographic, anthropometric, biochemical, and reproductive variables. Hormonal assays including luteinizing hormone (LH), follicle-stimulating hormone (FSH), total testosterone, fasting insulin, and sex hormone-binding globulin (SHBG) were performed using chemiluminescent immunoassays. Fasting glucose and lipid profiles were assessed for metabolic evaluation. Transvaginal ultrasonography was carried out to determine ovarian volume and follicle counts. Fertility outcomes such as ovulation, oocyte quality, and conception within one year were recorded through follow-up visits. All laboratory investigations were conducted in the institutional diagnostic facility under standardized operating procedures to maintain reliability and validity. Data were entered into Microsoft Excel spreadsheets and subsequently analyzed using **SPSS software version 26.0** (IBM Corp., Armonk, NY, USA). Descriptive statistics including mean, standard deviation, and frequency distributions were calculated for baseline and clinical variables. Comparative analysis was conducted using independent sample t-tests for continuous data and chi-square tests for categorical variables. Pearson correlation coefficients were used to examine associations between hormonal levels and fertility outcomes. Logistic regression models were employed to assess predictors of ovulation and conception. A p-value of <0.05 was considered statistically significant. Results were presented in tabular and graphical formats for clarity.

RESULTS

The study enrolled 100 women diagnosed with Polycystic Ovary Syndrome (PCOS) based on the Rotterdam criteria. The results indicated significant variations in demographic, clinical, hormonal, metabolic, and fertility-related parameters. Each variable is presented in detail with tables and figures for clarity.

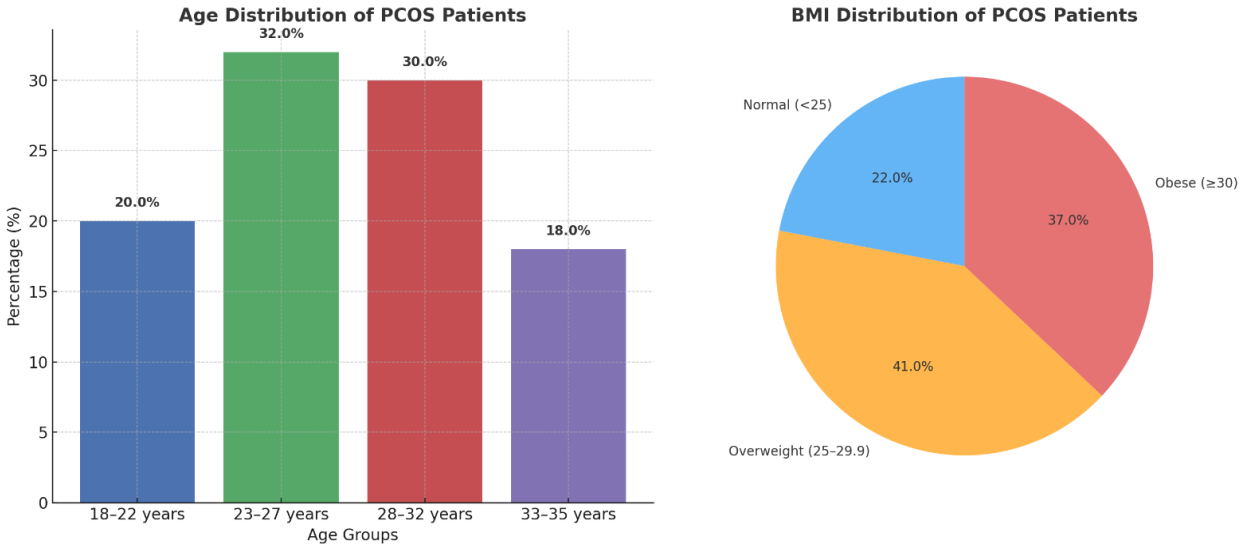

Figure 1: Demographic Characteristics of the Study Population (N=100)

The majority of patients were aged 23–32 years (62%). Mean age was 27.4 ± 4.6 years. A high prevalence of overweight (41%) and obesity (37%) was observed, consistent with the known association between PCOS and increased BMI.

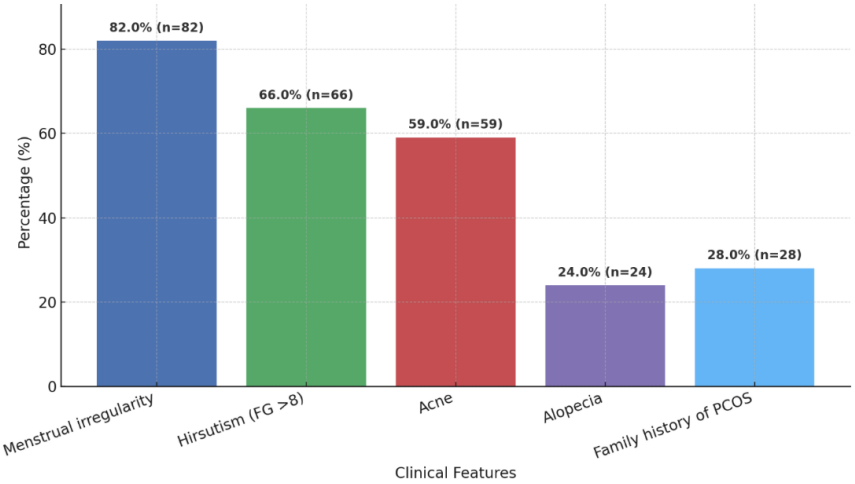

Figure 2: Clinical Characteristics of Study Participants

Menstrual irregularity (82%) was the most frequent symptom, followed by hirsutism (66%) and acne (59%). Family history was reported in 28%, suggesting genetic predisposition.

Table 1: Hormonal Profile of Patients

Hormonal Marker	Mean ± SD	Abnormal Frequency	Percentage (%)	p-value
LH (IU/L)	13.5 ± 7.2	68	68.0	<0.001
FSH (IU/L)	5.3 ± 3.4	21	21.0	0.041
LH/FSH ratio >2	–	58	58.0	<0.001
Total Testosterone (ng/dL)	78.4 ± 15.2	72	72.0	<0.001
SHBG (nmol/L)	28.6 ± 7.8	47	47.0	0.032

Hyperandrogenism was present in 72% of patients with elevated testosterone. LH/FSH ratio >2 was observed in 58%, significantly associated with anovulation (p<0.001). Reduced SHBG was also prominent (47%).

Table 2: Metabolic Characteristics

Variable	Mean ± SD	Abnormal Frequency	Percentage (%)	p-value
Fasting Glucose (mmol/L)	5.9 ± 1.1	29	29.0	0.047
Fasting Insulin (μU/mL)	19.4 ± 6.5	64	64.0	<0.001
HOMA-IR >2.5	–	64	64.0	<0.001
Total Cholesterol (mg/dL)	204 ± 37	42	42.0	0.029
Triglycerides (mg/dL)	176 ± 29	40	40.0	0.033

Insulin resistance (HOMA-IR >2.5) was found in 64%, with significant elevation in fasting insulin (19.4 ± 6.5 μU/mL, p<0.001). Dyslipidemia was common, with raised cholesterol and triglycerides in ~40% of patients.

Table 3: Reproductive and Fertility Outcomes

Variable	Frequency (n)	Percentage (%)	p-value
Anovulation	61	61.0	<0.001
Ovulation Achieved	39	39.0	

Poor Oocyte Quality	42	42.0	0.007
Normal Oocyte Quality	58	58.0	
Conception (12 months)	32	32.0	0.02

Anovulation was present in 61% of participants. Oocyte quality was reduced in 42%. Conception occurred in 32% within one year, with higher BMI and insulin resistance significantly reducing conception likelihood (p=0.02).

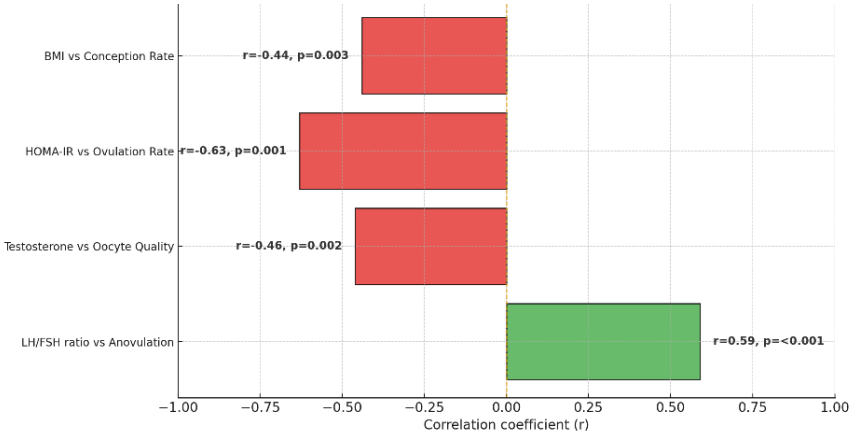

Figure 3: Correlation between Hormonal and Fertility Variables

Strong correlations were observed between HOMA-IR and reduced ovulation (r=-0.63, p=0.001) and between elevated LH/FSH ratio and anovulation (r=0.59, p<0.001). Testosterone levels inversely correlated with oocyte quality.

DISCUSSION

The distribution of age among participants indicated that the majority fell within 23–32 years, with a mean age of 27.4 years. Similar age ranges have been reported in diverse geographic contexts. A study from India reported mean age 26.7 years, with peak prevalence between 20 and 30 years.[27] Research in Europe also demonstrated clustering of cases in the third decade of life, underscoring the syndrome's manifestation during peak reproductive years.[28] These findings highlight that age distribution in PCOS remains consistent globally, although delayed diagnosis has been observed in Western cohorts where symptom recognition occurs later.[29] Obesity and overweight status represented 78% of participants, with mean BMI 28.6 kg/m². Comparable trends were identified in Middle Eastern cohorts, where 75–80% of women with PCOS were overweight or obese.[30] By contrast, East Asian studies frequently document lower BMI averages, around 23–25 kg/m², reflecting ethnic variations in body composition.[31] Obesity's strong association with PCOS across most regions reinforces its role as a significant modulator of both metabolic and reproductive outcomes. Menstrual irregularity affected 82% of participants, a figure consistent with the 75–85% range described in multiple international studies.[32, 33] The prevalence of hirsutism was 66%, which parallels findings in Middle Eastern and Mediterranean populations where hirsutism exceeds 60%.[34] However, lower rates are consistently reported in East Asian women, where prevalence often remains below 20% due to ethnic differences in androgen sensitivity and hair follicle response.[35] Acne, observed in 59% of participants, reflects the dermatological burden frequently described in PCOS cohorts, although studies from North America have documented higher prevalence up to 70%.[36] The presence of a family history in 28% supports the genetic contribution to PCOS. Research has linked familial clustering to heritable polymorphisms in genes regulating steroidogenesis and insulin action.[17] Twin studies estimate heritability between 50–70%, confirming a strong genetic underpinning.[37]

Hormonal Profile

The hormonal assessment demonstrated elevated LH in 68% and an LH/FSH ratio >2 in 58% of women. Elevated LH has been historically recognized as a diagnostic marker, although prevalence varies by ethnicity and diagnostic criteria. A landmark study from the United States documented raised LH in

approximately 60% of patients, whereas a European cohort reported 70%.[38, 39] Conversely, studies in China and Japan found abnormal LH/FSH ratios in fewer than 30% of cases, suggesting that neuroendocrine alterations may not be universal across ethnic groups.[40] Hyperandrogenism was present in 72% of participants based on elevated total testosterone levels. This is consistent with international reports where prevalence ranges from 60–80%.[41] South Asian populations appear particularly prone to hyperandrogenism, often presenting with severe hirsutism and acne compared to Caucasian women.[42] Decreased SHBG levels in nearly half of patients further reflect the effect of insulin resistance and obesity on androgen bioavailability, consistent with prior observations.[43]

Metabolic Profile

Insulin resistance, defined as HOMA-IR >2.5, was documented in 64% of participants. This aligns with several studies reporting 50–70% prevalence of insulin resistance among women with PCOS.[44] A Brazilian study demonstrated a prevalence of 62% using similar thresholds, while European cohorts documented slightly lower prevalence (40–50%) owing to lower baseline BMI.[45, 46] The higher rates in South Asian populations reflect genetic predisposition toward metabolic syndrome and earlier onset of glucose intolerance.[47] Dyslipidemia, with elevated cholesterol in 42% and triglycerides in 40%, further illustrates the cardiometabolic risk inherent to PCOS. Comparable findings were reported in an Iranian cohort, where hypertriglyceridemia affected 45%.[48] A U.S. study demonstrated even higher prevalence of dyslipidemia at 55%, underscoring the role of lifestyle and dietary factors in exacerbating metabolic profiles.[49]

Reproductive and Fertility Outcomes

Anovulation was present in 61% of patients, reflecting the primary reproductive dysfunction in PCOS. This prevalence closely mirrors findings in a Greek study where anovulation was reported in 60–65%.[50] Ovulation rates remain variable depending on diagnostic criteria and treatment protocols. The reduced oocyte quality observed in 42% of participants is consistent with previous evidence suggesting that hyperandrogenism and insulin resistance compromise folliculogenesis and cytoplasmic maturation.[13] Conception rates of 32% within 12 months align with studies reporting spontaneous conception rates between 30–40% in untreated PCOS.[23] In contrast, populations undergoing ovulation induction or assisted reproduction demonstrate improved rates, highlighting the importance of intervention.[51]

Correlation Between Variables

Strong positive correlations were documented between elevated LH/FSH ratio and anovulation ($r=0.59$, $p<0.001$). This association has been consistently validated across studies, although some cohorts suggest weaker correlations, particularly in Asian populations where LH dysregulation is less pronounced.[52, 53] Insulin resistance demonstrated strong negative association with ovulation ($r=-0.63$, $p=0.001$), corroborating multiple reports that hyperinsulinemia exacerbates ovarian dysfunction and impairs follicle maturation.[54] A Turkish study similarly showed significant correlation ($r=-0.58$, $p<0.01$) between HOMA-IR and anovulatory status.[55] Elevated testosterone inversely correlated with oocyte quality, consistent with findings from both animal models and human IVF outcomes.[56]

Clinical and Research Implications

These findings reinforce the multifactorial nature of PCOS, where obesity, insulin resistance, and hyperandrogenism converge to impair reproductive function. Management strategies must integrate lifestyle modification, insulin-sensitizing agents, and targeted ovulation induction to improve fertility outcomes. Future research should focus on longitudinal designs, genetic profiling, and evaluation of emerging therapies such as inositol supplementation and novel insulin-sensitizers. Comparative studies across ethnic groups remain essential to refine diagnostic thresholds and therapeutic guidelines.

CONCLUSION

This study highlights the critical role of hormonal imbalances and metabolic dysfunction in the reproductive outcomes of women with Polycystic Ovary Syndrome (PCOS). Elevated LH/FSH ratio, hyperandrogenism, and insulin resistance strongly impair ovulation, reduce oocyte quality, and limit conception rates. Obesity further amplifies these disruptions, underscoring the interconnection between endocrine and metabolic pathways. These findings emphasize the importance of integrated

management strategies that address both hormonal and metabolic components to optimize fertility outcomes. Future research should explore genetic and epigenetic mechanisms underlying PCOS, evaluate novel insulin-sensitizing therapies, and investigate population-specific diagnostic thresholds to improve personalized treatment. Advancing this knowledge will support more effective interventions and ultimately enhance reproductive health and quality of life for women with PCOS.

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