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Original Research Article

Hormonal Dysregulation and Its Metabolic Correlates in Polycystic Ovary Syndrome: A Case-Control Study of LH, FSH and Insulin Resistance

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Abstract

Polycystic ovary syndrome (PCOS) is a complex endocrine metabolic disorder characterized by chronic anovulation, hyperandrogenism, and insulin resistance, affecting women of reproductive age worldwide. This study aimed to evaluate key hormonal and metabolic alterations in women with PCOS by comparing luteinizing hormone (LH), follicle-stimulating hormone (FSH), and insulin resistance (HOMA-IR) between affected women and healthy controls. A total of 400 women aged 18-45 years were enrolled in a hospital-based case control design, including 200 clinically diagnosed PCOS cases and 200 age-matched controls. Fasting blood samples were collected and analyzed for LH, FSH, fasting glucose, and fasting insulin using standardized chemiluminescent immunoassays, and insulin resistance was calculated using the HOMA-IR formula. The results demonstrated significantly elevated LH levels in PCOS subjects compared with controls, indicating altered hypothalamic pituitary regulation. FSH levels also differed significantly between the groups, reflecting possible population-specific endocrine variations. HOMA-IR values were markedly higher in the PCOS group, confirming substantial insulin resistance as a key metabolic feature of the disorder. Correlation analysis showed a positive association between LH and HOMA-IR and a negative association between FSH and HOMA-IR, highlighting the strong interaction between hormonal imbalance and metabolic dysfunction. These findings reinforce PCOS as a multifaceted condition driven by interconnected endocrine and metabolic mechanisms. The study emphasizes that early recognition of hormonal abnormalities and insulin resistance is essential for improving reproductive outcomes, preventing long-term metabolic complications, and guiding comprehensive clinical management in women with PCOS.

Keywords: PCOS, HOMA-IR, FSH, LH.

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Introduction

Polycystic ovary syndrome (PCOS) is a prevalent and multifaceted endocrine disorder affecting women of reproductive age, characterized by reproductive, metabolic, and hormonal disturbances [1]. Once considered primarily a gynecological condition, PCOS is now recognized as a complex syndrome involving tightly interconnected neuroendocrine, metabolic, and ovarian pathways [2]. A central feature of its pathophysiology is disruption of the hypothalamic-pituitary-ovarian (HPO) axis [3].

In PCOS, increased GnRH pulse frequency disproportionately elevates luteinizing hormone (LH) while reducing follicle-stimulating hormone (FSH), resulting in excessive androgen production by theca cells and impaired follicular maturation [4]. This leads to chronic anovulation and menstrual irregularities. Although the LH/FSH ratio is not used diagnostically due to population variability, elevated LH remains a marker of neuroendocrine dysfunction in many women [5]. Metabolic dysfunction, particularly insulin resistance (IR), is another core feature [6]. Reduced cellular response to insulin triggers compensatory hyperinsulinemia, which amplifies ovarian androgen synthesis, suppresses sex hormone-binding globulin (SHBG), and interacts with LH to exacerbate hormonal imbalance [7]. Insulin resistance occurs even in lean women with PCOS, indicating intrinsic metabolic impairment, while coexisting obesity further worsens endocrine and metabolic disturbances [8].

Emerging research highlights additional regulators linking metabolism and reproduction. Neuroendocrine peptides such as kisspeptin, neurokinin B, and dynorphin modulate GnRH pulsatility [9]. Whereas adipokines like adiponectin and leptin influence insulin sensitivity, inflammation, and ovarian steroidogenesis [10]. These factors underscore the complex interplay between endocrine and metabolic pathways in PCOS. Evaluating hormonal markers (LH, FSH) alongside metabolic indicators (fasting insulin, HOMA-IR) provides a comprehensive understanding of PCOS heterogeneity. The present study therefore aims to assess LH, FSH, and HOMA-IR levels in women with PCOS and explore their interrelationships to better understand endocrine-metabolic interactions and inform targeted therapeutic strategies.

Methodology

A hospital-based cross-sectional case-control study was conducted on 400 women aged 18-45 years, including 200 clinically diagnosed PCOS patients and 200 age-matched healthy controls [11]. PCOS diagnosis was based on Rotterdam 2003 criteria, requiring at least two of the following: oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology confirmed by ultrasound [12]. Women with pregnancy, diabetes, thyroid disorders, androgen-secreting tumors, Cushing's syndrome, chronic illness, or oral contraceptive use were excluded. After an overnight fast of 8-10 hours, 5 mL of venous blood was collected between 8:00-10:00 AM. Serum LH and FSH were measured using chemiluminescence immunoassay, fasting glucose by glucose oxidase-peroxidase method, and fasting insulin by ELISA [13]. Insulin resistance was calculated using HOMA-IR. Data were expressed as mean ± SD, group differences assessed by Student's t-test, and correlations between hormonal and metabolic parameters analyzed using Pearson's coefficient. A p-value <0.05 was considered statistically significant.

Results

A total of 400 women (200 PCOS, 200 controls) were studied, with mean ages of 25.9 ± 4.8 and 26.3 ± 4.5 years, respectively, showing no significant difference. PCOS women exhibited significantly higher serum LH (43.3 ± 31.87 vs. 31.1 ± 28.9 mIU/mL) and FSH (59.9 ± 66.2 vs. 33.3 ± 36.35 mIU/mL) levels than controls ($p < 0.05$), reflecting hypothalamic–pituitary axis disturbances. HOMA-IR was markedly elevated in PCOS (3.76 ± 0.75 vs. 1.78 ± 0.43, $p < 0.05$), indicating insulin resistance and compensatory hyperinsulinemia. The mean values, standard deviations, and p-values for all parameters will be shown in (Table 1) and corresponding graphs representing differences between groups will be added as (Figures 1-3).

Table 1: Comparison of LH, FSH & HOMA IR Mean and standard deviation of PCOS patients with control

Parameters	Cases(mean+STD) n=200	control(mean+STD) n=200	t value	p value	Significance
LH	43.3±31.87	31.1±28.9	32.45771	< .05	significant
FSH	59.9±66.20	33.3±36.35	4.96934	< .05	significant
IR	3.76±0.75	1.78±0.43	32.45771	< .05	significant

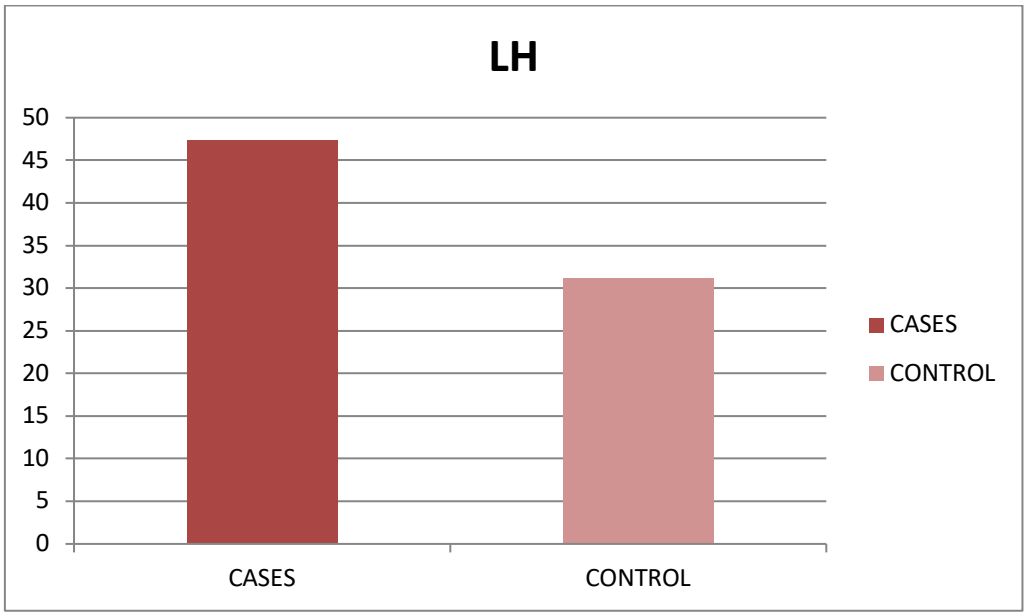

Figure 1: Distribution of cases and controls based on LH levels

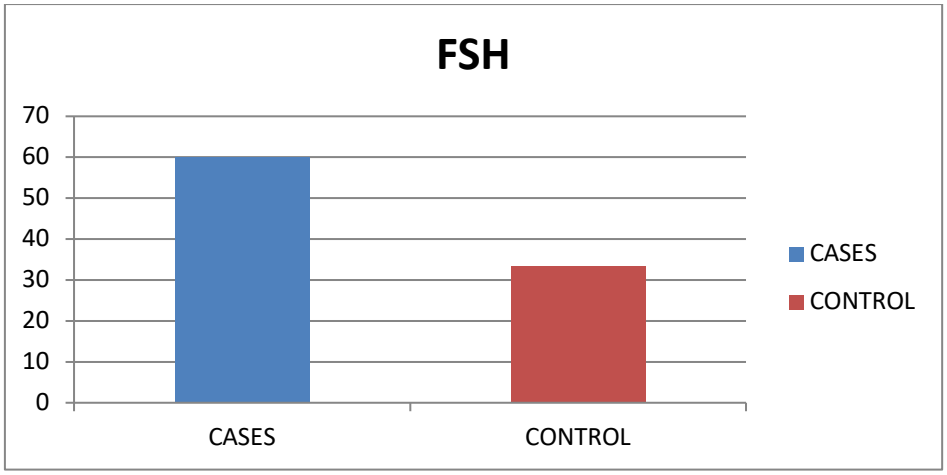

Figure 2: Distribution of cases and controls based on FSH levels

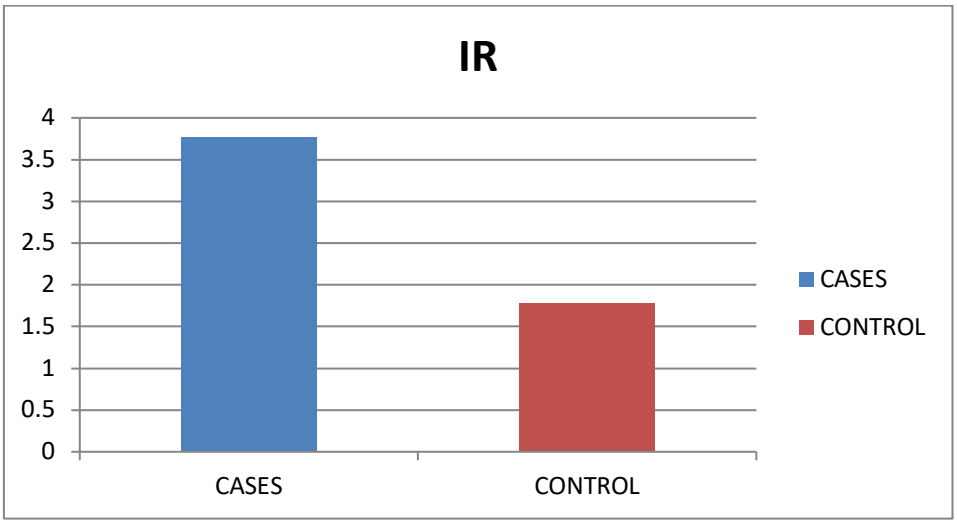

Figure 3: Distribution of cases and controls based on HOMA IR levels

Correlation Analysis between Hormonal and Metabolic Parameters: Pearson correlation analysis in PCOS women showed a positive association between LH and HOMA-IR, indicating higher LH levels correspond with increased insulin resistance. Conversely, FSH negatively correlated with HOMA-IR, suggesting that lower FSH levels are linked to greater metabolic dysfunction, reflecting the interplay between gonadotropin imbalance and insulin resistance (Figure 4 and 5).

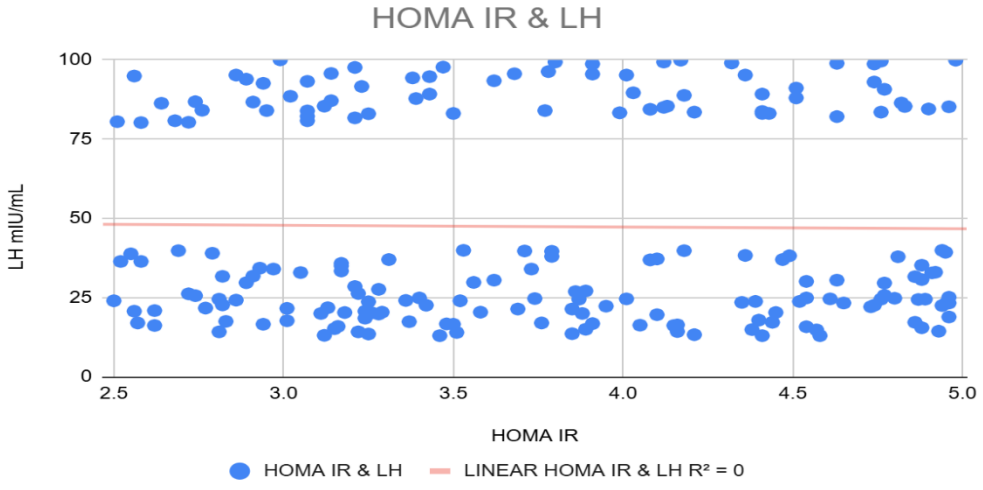

Figure 4: Graph showing Pearson's correlation of HOMA IR & LH in cases

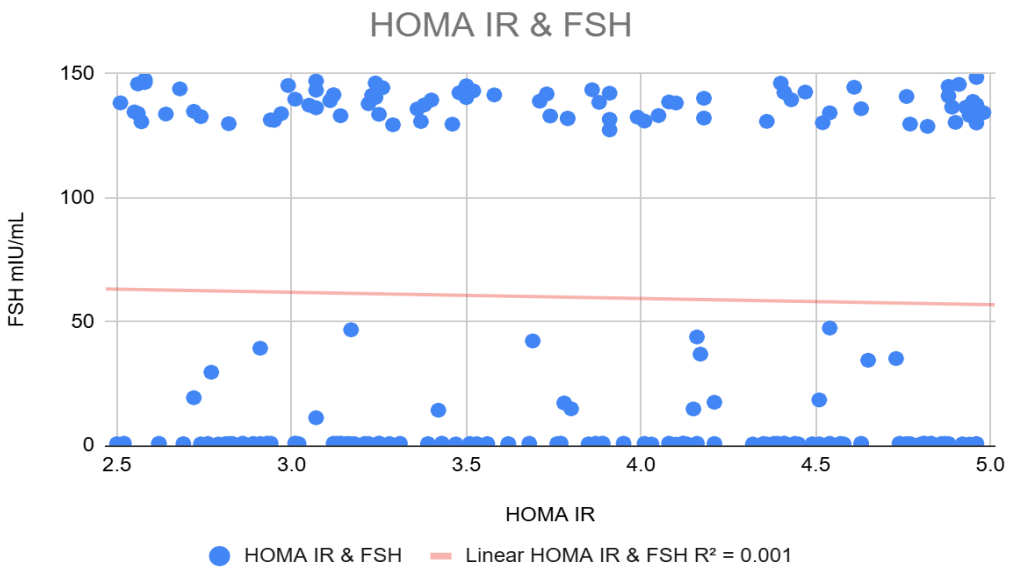

Figure 5: Graph showing Pearson's correlation of HOMA IR & FSH in cases

Discussion

The present case control study was conducted to investigate key hormonal and metabolic alterations in women with polycystic ovary syndrome by comparing serum LH, FSH, and HOMA-IR values between 200 PCOS cases and 200 healthy controls using standardized biochemical methods, and the findings strongly reinforce the multifactorial nature of PCOS [14]. The significantly elevated LH levels observed among PCOS subjects reflect a fundamental disturbance in hypothalamic pituitary regulation, where increased GnRH pulsatility preferentially enhances LH secretion, promoting excessive androgen synthesis and contributing to chronic anovulation [15].

Although classical descriptions of PCOS often report normal or marginally reduced FSH levels, the markedly higher FSH concentrations in this dataset suggest population-specific endocrine variation or a compensatory pituitary response attempting to overcome follicular arrest, emphasizing that gonadotropin expression in PCOS can vary across ethnic and metabolic backgrounds [16]. The pronounced elevation in HOMA-IR among PCOS women, which in our study was nearly double that of controls, indicates substantial insulin resistance a core metabolic abnormality in PCOS that exacerbates endocrine dysfunction by stimulating ovarian theca cells, reducing hepatic SHBG synthesis, and amplifying hyperandrogenism [17]. These metabolic endocrine interactions are further supported by the correlation analysis, which revealed a positive association between LH and HOMA-IR and a negative association between FSH and HOMA-IR, demonstrating that increased insulin resistance parallels worsening gonadotropin imbalance. Such correlations highlight the bidirectional relationship in which hormonal dysregulation influences metabolic pathways, while metabolic disturbances simultaneously aggravate reproductive endocrine abnormalities. These findings align with emerging evidence that PCOS should not be viewed purely as a reproductive disorder but rather as a systemic condition marked by intertwined metabolic and hormonal dysfunction. The methodology applied in this study including strict inclusion and exclusion criteria, fasting biochemical assessment, and validated immunoassay techniques ensures reliable comparison between groups and supports the conclusion that simultaneous evaluation of endocrine markers and insulin resistance provides a more comprehensive picture of PCOS pathophysiology [18]. Although the cross-sectional design limits causal interpretation, the large sample size and clear statistical significance strengthen the validity of the results and emphasize the clinical importance of routinely assessing both hormonal and metabolic parameters in women with PCOS (19). Overall, this study demonstrates that elevated LH, altered FSH, and significantly increased HOMA-IR collectively characterize the endocrine metabolic phenotype of PCOS, underscoring the need for early identification and integrated treatment approaches that address both hormonal imbalance and insulin resistance to improve reproductive outcomes, metabolic health, and long-term disease prevention in affected women.

Conclusion

The findings of this case control study demonstrate that women with polycystic ovary syndrome exhibit significant disturbances in both hormonal and metabolic regulation, as evidenced by markedly elevated LH, altered FSH concentrations,

and substantially increased HOMA-IR values compared with healthy controls. These results highlight the interconnected nature of endocrine and metabolic dysfunction in PCOS, where insulin resistance amplifies gonadotropin imbalance and contributes to the persistence of anovulation and hyperandrogenism. The significant correlations observed between LH, FSH, and HOMA-IR further reinforce PCOS as a multifaceted disorder driven by mutually reinforcing hormonal and metabolic pathways. Early recognition of these abnormalities is essential for improving clinical outcomes, as timely interventions targeting both insulin resistance and hormonal imbalance may enhance reproductive function, reduce long-term metabolic risks, and support overall health in affected women. This study underscores the importance of integrating metabolic assessment into routine PCOS evaluation and encourages a comprehensive approach to diagnosis and management.

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