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Association of serum ferritin and insulin resistance in PCOD patients

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ABSTRACT
Background:

Polycystic Ovary Syndrome (PCOS) is a common endocrine disorder associated with insulin resistance and metabolic abnormalities. Emerging evidence suggests that altered iron metabolism, reflected by elevated serum ferritin levels, may contribute to insulin resistance and related complications in PCOS.

Objective:

To evaluate serum ferritin levels and insulin resistance in women with PCOS and compare them with age- and BMI-matched healthy controls.

Materials and Methods

This hospital-based case–control study was conducted at Lady Hardinge Medical College, New Delhi, over a period of two years (February 2021 to September 2022). A total of 60 subjects were included, comprising 30 diagnosed cases of PCOS (based on Rotterdam criteria) and 30 healthy controls matched for age and BMI. Fasting venous blood samples were collected and analyzed for serum ferritin, insulin, fasting blood glucose, HbA1c, and serum iron using standard biochemical methods. Insulin resistance was assessed using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR). Statistical analysis was performed using Student's t-test, Chi-square test, and Pearson correlation, with $p<0.05$ considered significant.

Results

The mean serum insulin and HOMA-IR levels were significantly higher in PCOS cases compared to controls ($p<0.00001$). Serum ferritin levels were also significantly elevated in PCOS subjects (81.50±63.98 ng/mL) compared to controls (50.83±39.92 ng/mL) ($p=0.029$). Fasting blood glucose and HbA1c levels were significantly higher in cases ($p<0.001$). A positive correlation was observed between serum ferritin and insulin resistance, fasting blood glucose, and insulin levels, while a negative correlation was noted with HbA1c.

Conclusion

Elevated serum ferritin is associated with insulin resistance in PCOS, suggesting its role as a potential biomarker for metabolic risk assessment

Keywords

Polycystic Ovary Syndrome, Serum Ferritin, Insulin Resistance, HOMA-IR, Iron Metabolism

Introduction:

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine disorders, affecting approximately 5–10% of women in the reproductive age group. It is a heterogeneous condition characterized by chronic anovulation and hyperandrogenism. In addition to reproductive

abnormalities, metabolic disturbances such as obesity and insulin resistance are frequently associated with PCOS (1,2).

Insulin resistance is a key feature in the pathophysiology of PCOS and is an established risk factor for the development of type 2 diabetes mellitus and cardiovascular diseases (3). Recent evidence suggests that altered iron metabolism may contribute to insulin resistance at the cellular level. Increased body iron stores, reflected by elevated serum ferritin levels, have been implicated in insulin-resistant states such as T2DM and metabolic syndrome (4).

The relationship between iron metabolism and insulin resistance appears to be bidirectional. Iron overload may impair pancreatic β-cell function and worsen insulin sensitivity, while hyperinsulinemia may enhance intestinal iron absorption by upregulating hypoxia-inducible factor-1α, thereby increasing body iron stores (5). Furthermore, hyperinsulinemia in PCOS contributes to increased androgen production, predisposing individuals to long-term metabolic complications, including impaired glucose tolerance, T2DM, and cardiovascular diseases (6,7).

Therefore, the present study was designed to evaluate serum ferritin levels and insulin resistance in women with PCOS and to compare them with age- and BMI-matched healthy controls.

MATERIALS AND METHODS

Study Design and Setting

This hospital-based case–control study was conducted in the Departments of Biochemistry and Obstetrics & Gynaecology at Lady Hardinge Medical College and associated Kalawati Saran Hospital, New Delhi, India. The study was carried out over a period of two years from February 2021 to September 2022. The study protocol was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants before inclusion in the study. The study included a total of 60 subjects, comprising 30 diagnosed cases of Polycystic Ovary Syndrome (PCOS) and 30 age- and body mass index (BMI)-matched healthy controls. Females aged between 15 and 45 years, diagnosed cases of PCOS based on Rotterdam criteria, confirmed by ultrasonography, were included in the study. Secondary causes of hyperandrogenism, diabetes

mellitus, hypertension, or other chronic systemic illnesses, and Pregnant women were excluded from the study.

Sample Collection and analysis:

Approximately 5 mL of venous blood was collected from each subject under aseptic conditions from the median cubital vein after overnight fasting. The blood samples were allowed to clot for 30 minutes and then centrifuged to separate serum. Serum samples were stored at −80°C until analysis. The following biochemical parameters were measured: Serum ferritin and insulin by Electrochemiluminescence immunoassay, Serum iron by Ferrozine method, fasting blood glucose by Hexokinase method, Glycated hemoglobin (HbA1c): Cation-exchange high-performance liquid chromatography (HPLC), and Fasting Insulin. Insulin resistance was calculated using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) using the formula:

$$HOMA\text{-}IR = \frac{\text{Fasting insulin (μU/mL)} \times \text{Fasting glucose (mg/dL)}}{405}$$

Statistical Analysis

Data were analyzed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were expressed as frequency and percentage. Student's independent t-test, Chi-square test/Fisher's exact test were applied. A p-value <0.05 was considered statistically significant.

RESULTS

The present case–control study included a total of 60 subjects, comprising 30 women diagnosed with PCOS and 30 age-matched healthy controls. The mean age of cases and controls was 26.26±5.77 years and 28.73±5.95 years, respectively, showing no statistically significant difference between the groups.

Table 1 revealed that the mean age between the two groups was comparable and not statistically significant. However, serum insulin levels and HOMA-IR were significantly higher in PCOS cases, indicating marked insulin resistance ($p<0.00001$). Serum ferritin levels were also significantly elevated in cases compared to controls ($p=0.029$). In addition, fasting blood glucose

and HbA1c levels were significantly higher in the PCOS group ($p<0.001$). Although serum iron levels were higher in cases, the difference was not statistically significant ($p>0.05$).

Table 1: Comparison of Baseline and Biochemical Parameters between Cases and Controls

Parameter	Cases (Mean ± SD)	Controls (Mean ± SD)	p-value
Age (years)	26.26 ± 5.77	28.73 ± 5.95	NS
Insulin (µU/mL)	37.61 ± 23.03	13.63 ± 7.07	<0.00001*
HOMA-IR	11.25 ± 7.45	3.02 ± 1.90	<0.00001*
Ferritin (ng/mL)	81.50 ± 63.98	50.83 ± 39.92	0.029*
Fasting Blood Glucose (mg/dL)	119.43 ± 35.00	90.78 ± 16.90	0.000161*
HbA1c (%)	5.74 ± 0.64	4.99 ± 0.57	0.000011*
Serum Iron (µg/dL)	108.26 ± 32.57	96.83 ± 37.50	0.212

****Statistically significant ($p<0.05$); NS: Not Significant

Table 2 observed that a majority of PCOS cases (73.3%) exhibited severe insulin resistance (HOMA-IR >5), whereas only 10% of controls fell into this category. In contrast, more than half of the controls (53.3%) had normal insulin resistance (HOMA-IR <3), compared to only 3.3% of cases. This difference in distribution was statistically highly significant ($p<0.00001$), indicating a strong association between PCOS and increased insulin resistance.

Table 2: Distribution of Insulin Resistance (HOMA-IR) among Cases and Controls

HOMA-IR Category	Cases (n=30)	Controls (n=30)	p-value
<3 (Normal)	1 (3.3%)	16 (53.3%)	<0.00001*
3–5 (Moderate)	7 (23.3%)	11 (36.7%)	

>5 (Severe)	22 (73.3%)	3 (10%)	
Mean ±SD	11.254±7.45	3.029±1.90	

Table 3 demonstrates that serum ferritin demonstrated a significant positive correlation with fasting blood glucose, serum insulin, and HOMA-IR ($p<0.05$), indicating its association with insulin resistance and glycemic status. A negative correlation was observed between serum ferritin and HbA1c, which was statistically significant. Although serum ferritin showed a positive correlation with serum iron, this association was not statistically significant ($p>0.05$).

Table 3: Correlation of Serum Ferritin with Biochemical Parameters in PCOS Cases

Parameter	**Correlation Coefficient (r)**	**p-value**
Fasting Blood Glucose	0.0834	0.000161*
Serum Iron	0.2974	0.212
Insulin	0.166	<0.00001*
HOMA-IR	0.1586	<0.00001*
HbA1c	-0.1398	0.000011*

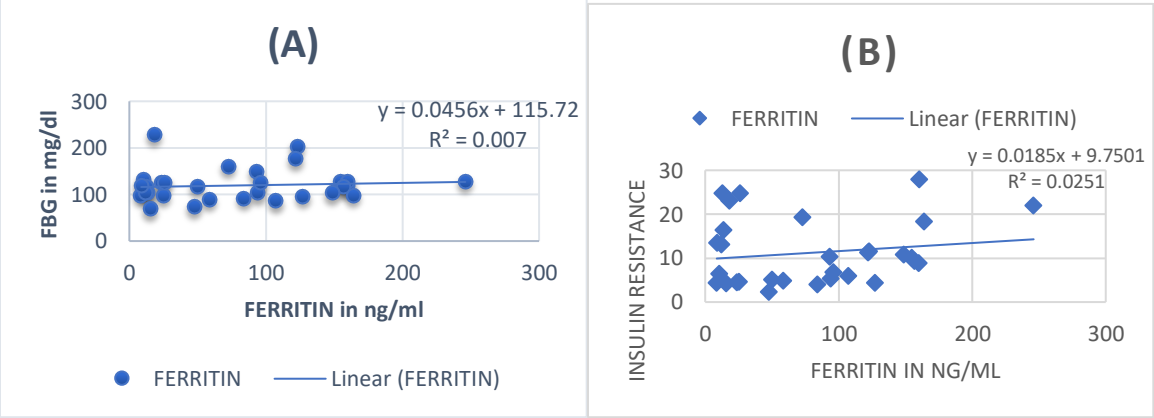


Fig 1 (A) scatter diagram of Serum Ferritin vs Fasting blood glucose (Cases and Controls)

(B) scatter diagram of Serum Ferritin vs HOMA-IR

Discussion:

The present study evaluated serum ferritin levels and insulin resistance in women with Polycystic Ovary Syndrome (PCOS) and compared them with age- and BMI-matched healthy controls. The findings demonstrated significantly higher levels of serum insulin, HOMA-IR, serum ferritin, fasting blood glucose, and HbA1c in PCOS cases, indicating the presence of insulin resistance and altered metabolic status. Insulin resistance is a well-recognized feature of PCOS and plays a central role in its pathogenesis. In the present study, significantly elevated fasting insulin levels and HOMA-IR values were observed in PCOS cases compared to controls. These findings are consistent with those reported by Dunaif A, who described insulin resistance as a key abnormality in PCOS, contributing to hyperandrogenism and metabolic dysfunction [8].

Serum ferritin levels were significantly higher in PCOS cases than in controls in this study. This observation is in agreement with studies by Luque-Ramirez M et al., who reported increased body iron stores in women with PCOS, suggesting that hyperinsulinemia may lead to increased intestinal iron absorption and reduced menstrual iron loss [5]. Similarly, Sharifi F et al. demonstrated elevated serum ferritin levels in PCOS subjects, supporting the association between iron overload and metabolic disturbances [9].

The present study also showed a significant positive correlation between serum ferritin and insulin resistance parameters (insulin and HOMA-IR), as well as fasting blood glucose. These findings suggest that increased iron stores may contribute to impaired glucose metabolism. Fernández-Real JM et al. reported that excess iron may lead to oxidative stress and pancreatic β-cell dysfunction, thereby promoting insulin resistance and diabetes [3].

Additionally, serum ferritin showed a negative correlation with HbA1c in the present study, although the exact mechanism remains unclear and warrants further investigation. Similar inconsistencies have been reported in previous studies, possibly due to differences in study populations and sample sizes. Although serum iron levels were higher in PCOS cases, the difference was not statistically significant, indicating that ferritin may be a more sensitive marker of iron status in relation to metabolic disturbances.

Overall, the findings of this study support the hypothesis that increased body iron stores are associated with insulin resistance in PCOS. The interplay between hyperinsulinemia and iron

metabolism appears to be bidirectional, where insulin resistance promotes iron accumulation, and excess iron further exacerbates metabolic dysfunction. Although the present study has certain limitations. The sample size was relatively small, and the study was conducted at a single center, which may limit the generalizability of the findings.

Conclusion: Women with Polycystic Ovary Syndrome show significantly higher serum ferritin and insulin resistance, with a positive correlation between them. Serum ferritin may serve as a simple biomarker for early metabolic risk assessment alongside Homeostasis Model Assessment of Insulin Resistance; however, larger longitudinal studies are needed to confirm its clinical utility and establish definitive cut-off values.

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Conflict of interest: None

Author Contribution: Dr. Sarita Choudhary contributed to the study concept and design, supervision of the research work. Dr. Ekta Mittal was involved in statistical analysis and drafting of the manuscript. Dr. Neeraj Yadav contributed to data interpretation and critical revision of the manuscript. Dr. Jaswant Kaur participated in manuscript writing and final editing of the manuscript. All authors reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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