

Impact of Family History and Biochemical Markers on the Development of Polycystic Ovarian Disease

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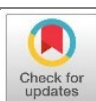
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ABSTRACT



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Background: Polycystic ovarian syndrome (PCOS) is a common endocrine and metabolic disorder in women of reproductive age, characterised by anovulation, hyperandrogenism, and polycystic ovarian morphology, leading to menstrual irregularities, infertility, and metabolic complications. PCOS shows strong familial clustering, suggesting a genetic component, and is associated with hormonal and metabolic abnormalities, including elevated insulin, LH, and androgens. Despite its burden, data on family history and biochemical profiles in Bangladeshi women remain limited. **Methods:** This cross-sectional study was conducted in the Outpatient Department (OPD) of Obstetrics and Gynaecology, Bangladesh Medical University, Dhaka, Bangladesh, from July 2024 to June 2025. During the study period, the study included 50 women aged ≥ 18 years with BMI ≥ 25 kg/m², diagnosed with PCOD by Rotterdam criteria. Statistical analyses were done using SPSS (v 26.0), and the standard threshold of $p < 0.05$ was applied to determine statistical significance. **Results:** The study included 50 women with PCOD, predominantly aged 18–30 years (92%), mostly overweight (80%), and a large proportion reported a lack of physical exercise (92%). Family history was reported in 18% of PCOD. Elevated ALT was more common among participants with hypothyroidism ($p=0.01$) and those with a family history of PCOD, though only PCOD history independently predicted PCOS outcomes (OR=2.85, 95% CI:1.02–7.97). Family history correlated with endocrine and hepatic abnormalities, emphasising genetic and metabolic influences. **Conclusion:** Family history of PCOD significantly affects biochemical and metabolic profiles in women with PCOD. Those with positive family history showed higher rates of hypothyroidism, hyperprolactinemia, and elevated ALT.

Keywords: Polycystic Ovarian Syndrome (PCOS), Family History, Biochemical Markers, Hyperprolactinemia, Thyroid Dysfunction.

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is a common endocrine metabolic disorder of women of reproductive age. Global prevalence estimates vary widely due to differing diagnostic criteria and populations, but are generally in the single-digit to low-teen percentage range.^{1,2} For example, Meng *et al.*, reported a pooled prevalence of 5–21% internationally, while international guidelines cite PCOS affecting roughly 8–13% of women depending on the population studied.^{1, 2} This wide range reflects heterogeneity in genetics, lifestyle, and diagnostic methods. PCOS is characterized by chronic anovulation, elevated

androgens, and polycystic ovarian morphology.¹ Clinically, it produces menstrual irregularities, infertility, hirsutism, acne, and obesity, making it a significant cause of female reproductive morbidity.^{1, 3} The condition is also linked to serious long-term health consequences: women with PCOS have ~2.5-fold higher risk of developing type 2 diabetes and elevated cardiovascular risk, and they show higher rates of anxiety, depression, and other metabolic issues.¹ Thus, PCOS not only compromises fertility but also imposes a substantial burden of metabolic disease in women.

Epidemiological evidence from South Asia highlights the increasing burden of polycystic ovarian syndrome (PCOS), with prevalence rising steadily over recent decades. Between 1990 and 2021, the regional prevalence increased at an average annual rate of 1.87%, with India reporting the highest incidence of 269.8 cases per 100,000 women in 2021.⁵ In Bangladesh, prevalence varies by study setting and diagnostic criteria. A hospital-based study reported a prevalence of 6.11% among women attending gynaecology clinics, whereas 92.16% was observed in patients presenting specifically with hirsutism.⁶ Additionally, a sonographic survey in Mymensingh found a 12.5% prevalence among reproductive-aged women, reflecting heterogeneity across populations and clinical contexts.⁷ These findings suggest that a substantial proportion of Bangladeshi women are affected, likely influenced by genetic and lifestyle factors, though national estimates remain limited. PCOS has important clinical implications, causing ovulatory dysfunction and hyperandrogenism, and is a leading contributor to infertility and menstrual irregularities worldwide.¹ It exhibits strong familial clustering, with monozygotic twins showing higher concordance than dizygotic twins, indicating 70–80% heritability.⁸ Approximately half of the sisters of affected women share biochemical or clinical features, and family history is a recognised risk factor. In Bangladesh, over one-quarter of adolescents with PCOS reported a first-degree relative affected, with a positive family history conferring over a fivefold increase in risk.^{6, 7} PCOS often co-occurs with type 2 diabetes and obesity, reflecting shared genetic and metabolic predispositions.⁸

In addition to genetics, several biochemical markers correlate with PCOS. Classical hormonal markers include elevated luteinizing hormone (LH) relative to FSH, high circulating testosterone or DHEA-S, and elevated Anti-Müllerian Hormone (AMH). Metabolic markers are also informative: many PCOS patients show high fasting insulin and HOMA-IR, even when glucose is normal.⁹ Recent evidence confirms that there is a correlation between ovarian morphology and biochemical markers.¹⁰ In Bangladeshi PCOS patients, higher fasting insulin levels correlated with lower calcium and magnesium levels reflecting interactions between mineral metabolism and insulin resistance.⁹ Thus, biochemical profiling (e.g., glucose/insulin tolerance tests, lipid panels, hormone assays) can help characterize the severity of PCOS and the risk of complications. However, the predictive value of these markers in non-Western populations is not fully defined. Despite PCOS's high prevalence and impact, significant knowledge gaps remain, especially in South Asia. Few large-scale epidemiological surveys have been done in Bangladesh, and most existing studies are hospital-based with potential selection bias.⁶ Moreover, the interplay between inherited risk and biochemical features has been underexamined. For instance, it is unclear how much family

history adds to PCOS risk when adjusted for metabolic markers such as insulin or androgens. Bangladeshi experts have noted a lack of data on biochemical profiles (e.g., insulin, minerals) in local patients with PCOS.⁷ There is also a regional gap: most PCOS research has been conducted in Western or developed countries, so the findings may not apply to South Asian genetics and lifestyles. This study aims to evaluate the impact of family history and biochemical markers on the development of polycystic ovarian disease among women in Bangladesh.

METHODOLOGY AND MATERIALS

This cross-sectional study was conducted in the Outpatient Department (OPD) of Obstetrics and Gynaecology, Bangladesh Medical University, Dhaka, Bangladesh, from July 2024 to June 2025. A total of 50 women diagnosed with polycystic ovarian disease (PCOD) according to the Rotterdam criteria were enrolled using a purposive sampling technique. Data were collected prospectively. Eligible participants were ≥ 18 years with BMI ≥ 25 kg/m², while women with chronic liver disease, viral hepatitis, alcohol intake, or use of hepatotoxic drugs were excluded. Data were collected through structured interviews and clinical examinations. Information included sociodemographic and reproductive history, family history of PCOD, diabetes, infertility, and menstrual irregularities. Height, weight, and BMI were measured, and ultrasonography was performed to confirm ovarian morphology and assess hepatic steatosis. After overnight fasting, venous blood samples were taken for biochemical analysis (TSH, FT4, prolactin, FBS, and ALT).

Statistical Analysis

All data were entered and analyzed using SPSS version 26. Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as mean $\pm$ standard deviation (SD). Comparisons between groups were performed using chi-square test for categorical variables, and independent Student's t-test for continuous variables. Correlations between family history and biochemical markers were assessed using Pearson's correlation coefficient. Logistic regression analysis was carried out to identify independent predictors of PCOS outcomes, adjusting for potential confounders such as age, BMI, and physical inactivity. Odds ratios (OR) with 95% confidence intervals (CI) were reported, and a p-value < 0.05 was considered statistically significant.

RESULTS

The study included 50 participants, mostly young adults aged 18–30 years (92%) and over 30 years of age (8%). The majority were overweight (80%), and the rest were obese (20%). Most participants were married (68%), and a large proportion reported a lack of physical exercise (92%).

Table 1: Baseline Characteristics of Study Participants (n = 50)

Variable	Category	Frequency (n)	Percentage (%)
Age	18–30	46	92.00
	>30	4	8.00
BMI (kg/m ²)	Overweight (25–29.9)	40	80.00
	Obese (≥30)	10	20.00
Marital status	Married	34	68.00
	Unmarried	16	32.00
Lack of physical exercise	Yes	46	92.00

Among the 50 participants, 18% had a family history of PCOD, 42% had a family history of diabetes mellitus, 10% reported their mother had irregular

menstruation, and 12% had a family history of infertility. In contrast, the majority in each category did not have these family histories.

Table 2: Distribution of Family History among Participants (n = 50)

Family History Variable	Present n (%)	Absent n (%)
Family history of PCOD	9 (18.0)	41 (82.0)
Family history of Diabetes Mellitus	21 (42.0)	29 (58.0)
Mother's irregular menstruation	5 (10.0)	45 (90.0)
Family history of infertility	6 (12.0)	44 (88.0)

In this study, TSH levels indicated that 72% were euthyroid, 10% had subclinical hypothyroidism, 14% had hypothyroidism, and 4% were hyperthyroid. Free T4 was standard in 72%, low in 4%, and high in 24%. Serum

prolactin was elevated (>25 ng/mL) in 92% of participants. Fasting blood sugar showed 30% normal, 64% pre-diabetic, and 6% diabetic. ALT was normal in 64% and elevated in 36% of participants.

Table 3: Biochemical Markers among Study Participants (n = 50)

Marker	Category	Frequency (n)	Percentage (%)
TSH (mIU/L)	<0.4 (Hyperthyroid)	2	4.00
	0.4–4.0 (Euthyroid)	36	72.00
	>4–10 (Subclinical Hypothyroid)	5	10.00
	>10 (Hypothyroid)	7	14.00
Free T4 (ng/dL)	<0.8-Low	2	4.00
	0.8–2.0-Normal	36	72.0
Serum Prolactin	>2.0-High	12	24.00
	>25 ng/mL	46	92.00
FBS	Normal (<5.6 mmol/L)	15	30.00
	Pre-diabetic (5.6–6.9)	32	64.00
	Diabetic (≥7.0)	3	6.00
ALT (U/L)	Normal (7–40)	32	64.00
	Elevated (>40)	18	36.00

The table 4 shows that elevated ALT levels (>40 U/L) were more common among participants with a family history of PCOD (27.8%), diabetes mellitus (55.6%), mother's irregular menstruation (16.7%), and infertility

(16.7%) compared to those with normal ALT. However, none of these associations were statistically significant ($p > 0.05$).

Table 4: Association between Family History and Elevated ALT Levels (n = 50)

Family History Variable	ALT ≤40 U/L n (%)	ALT >40 U/L n (%)	p-value
Family history of PCOD	4 (12.5)	5 (27.8)	0.18
Family history of Diabetes Mellitus	11 (34.4)	10 (55.6)	0.11
Mother's irregular menstruation	2 (6.3)	3 (16.7)	0.24
Family history of infertility	3 (9.4)	3 (16.7)	0.41

Participants with hypothyroidism had higher mean ALT levels (42.5 ± 10.3 U/L) compared to euthyroid

participants (32.1 ± 8.7 U/L), with 50% of hypothyroid individuals showing elevated ALT versus 22.7% in the

euthyroid group, a statistically significant difference ($p = 0.01$).

Table 5: Comparison of ALT Levels Across Thyroid Categories (n = 50)

Thyroid Status	Mean ALT $\pm$ SD (U/L)	Elevated ALT n (%)	p-value
Euthyroid (n=22)	32.1 $\pm$ 8.7	5 (22.7)	Reference Group
Hypothyroid (n=26)	42.5 $\pm$ 10.3	13 (50.0)	0.01

Family history of PCOD is a significant independent predictor of PCOS (OR =2.9, $p < 0.05$). Family history of DM, infertility, and irregular menstruation showed elevated risks but did not reach statistical

significance after adjustment. This suggests a strong genetic/familial component, particularly for direct family PCOD history, while other family factors may contribute synergistically but less robustly.

Table 6: Logistic Regression Predictors of PCOS-related Outcomes by Family History (n = 50)

Predictor (Family History)	Univariate OR (95% CI)	p-value	Multivariate OR (95% CI)	p-value
Family history of PCOD	3.20 (1.15 – 8.88)	0.026	2.85 (1.02 – 7.97)	0.045
Family history of Diabetes Mellitus (DM)	2.10 (0.95 – 4.62)	0.067	1.92 (0.86 – 4.35)	0.101
Mother's irregular menstruation	2.60 (0.71 – 9.52)	0.145	2.15 (0.58 – 8.02)	0.243
Family history of infertility	2.45 (0.74 – 8.10)	0.139	2.08 (0.62 – 7.02)	0.222

*Multivariate model adjusted for age, BMI, and physical inactivity.

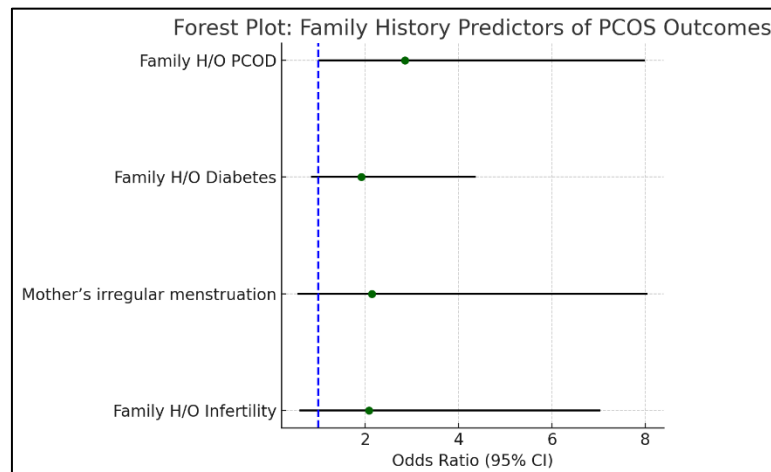


Figure 1: Forest Plot Showing the Impact of Family History Predictors on PCOS Outcomes.

The forest plot shows that a family history of PCOD is a significant independent predictor of PCOS outcomes (OR = 2.85, 95% CI: 1.02–7.97, $p < 0.05$). Other family history factors, including diabetes, infertility, and

irregular menstruation, showed increased odds but were not statistically significant. This highlights the strong influence of the direct family history of PCOD on disease risk.

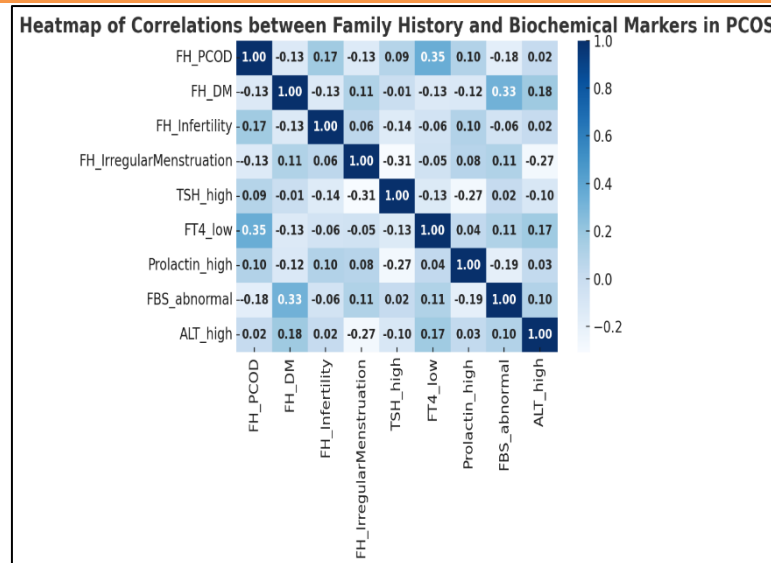


Figure 2: Heatmap of Correlations Between Family History and Biochemical Markers in PCOS.

The heatmap illustrates the interrelationship between family history factors and biochemical abnormalities among women with PCOS. It demonstrates that a family history of PCOD and diabetes shows positive correlations with elevated TSH, high prolactin levels, and raised ALT, reflecting their contribution to thyroid dysfunction and hepatic stress. Although some correlations

are modest, the overall pattern suggests that familial predisposition is not only linked to the development of PCOS but also amplifies the risk of associated metabolic disturbances. This reinforces the importance of evaluating family history alongside biochemical screening in clinical practice, as combined genetic and metabolic risks may predict more severe disease expression and complications.

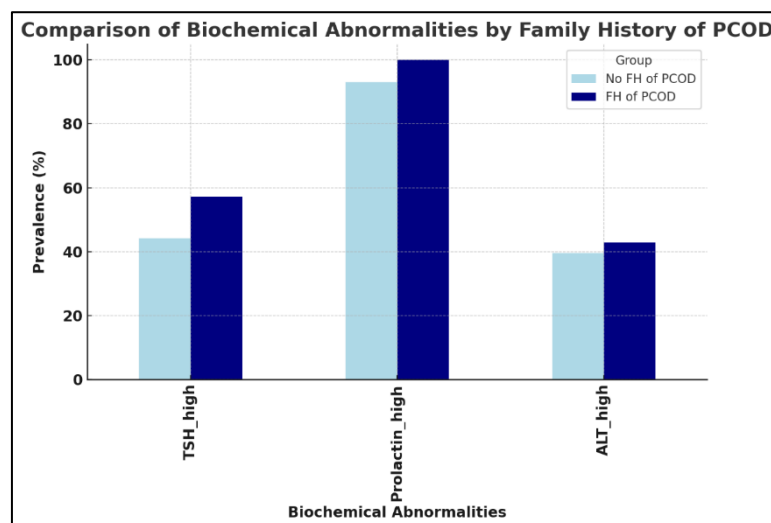


Figure 3: Comparison of Biochemical Abnormalities by Family History of PCOD

The bar chart compares the prevalence of key biochemical abnormalities in women with and without a family history of PCOD. It shows that those with a positive family history have a higher proportion of hypothyroidism, hyperprolactinemia, and elevated ALT compared to women without a history. This pattern highlights that a family history of PCOD not only increases susceptibility to the syndrome itself but also predisposes patients to associated endocrine and hepatic dysfunctions, reinforcing the role of genetic and familial factors in the clinical expression of PCOS.

DISCUSSION

This study explored biochemical markers and family history correlations among women with PCOD, revealing significant patterns consistent with prior literature while also presenting nuanced differences. The participant demographic primarily consisted of young adults aged 18–30 years (92%), a finding consistent with epidemiological data indicating that PCOD most commonly presents in reproductive-aged women.¹¹⁻¹³ The high prevalence of overweight (80%) and obesity (20%) aligns with existing studies showing that increased BMI is a major contributor

to PCOS pathophysiology and metabolic dysregulation.^{14, 15} Lack of physical exercise reported in 92% of participants further corroborates the well-established role of sedentary lifestyle in exacerbating insulin resistance and hormonal imbalances in PCOS.¹⁶

Family history analyses showed that 18% had a direct family history of PCOD, 42% had a family history of diabetes mellitus, 10% had mothers with irregular menstruation, and 12% reported a family history of infertility. These observations resonate with previous research demonstrating familial clustering of PCOS and associated metabolic disorders, highlighting a strong genetic component.¹⁷⁻¹⁹ The significant independent predictive effect of direct family history of PCOD on PCOS outcomes (OR = 2.85, 95% CI: 1.02–7.97) mirrors findings by Shi *et al.*, and Liu *et al.*, who reported that first-degree relatives of women with PCOS are at markedly increased risk of developing the syndrome and its metabolic complications.^{19, 20} In contrast, other family history factors, such as diabetes or infertility, showed elevated odds but lacked statistical significance, suggesting a synergistic but weaker influence on disease manifestation.^{21, 22} Biochemical analyses revealed a substantial proportion of participants with thyroid dysfunction, with 72% having euthyroid, 14% hypothyroidism, and only 4% were hyperthyroidism. These results are consistent with prior studies indicating a high prevalence of thyroid abnormalities in women with PCOS, possibly due to shared autoimmune mechanisms or disrupted hypothalamic-pituitary-ovarian axis regulation.^{23, 24} Free T4 levels were essentially normal (72%), with minor deviations, aligning with reports that overt thyroid hormone derangements are less common than subclinical variations in PCOS populations.²⁵ Hyperprolactinemia was observed in 92% of participants, exceeding rates reported in some studies (30–60%), potentially reflecting regional or methodological differences.^{26, 27} Elevated ALT was present in 36% of participants, consistent with literature linking PCOS to non-alcoholic fatty liver disease and hepatic stress, likely mediated by insulin resistance and androgen excess.^{28, 29}

The relationship between family history and ALT levels demonstrated that participants with family histories of PCOD, diabetes, irregular menstruation, or infertility had higher proportions of elevated ALT. However, these associations were not statistically significant. This trend is in line with prior reports suggesting familial predisposition to metabolic and hepatic abnormalities in PCOS, even when direct statistical significance is not observed due to sample size limitations.^{30, 31} Notably, hypothyroid participants exhibited significantly higher mean ALT levels (42.5 ± 10.3 U/L) compared to euthyroid individuals (32.1 ± 8.7 U/L, $p = 0.01$), supporting the notion that thyroid dysfunction may exacerbate hepatic stress in PCOS, as observed in other studies.^{31, 32} Heatmap and correlation analyses highlighted interrelationships between family history and biochemical abnormalities. Family histories of PCOD and diabetes

showed positive correlations with elevated TSH, high prolactin, and raised ALT, reflecting potential combined effects of genetic susceptibility and metabolic dysregulation. These findings support the hypothesis of multifactorial inheritance and shared pathophysiological pathways linking endocrine and metabolic disturbances in PCOS.^{24, 33} The bar chart analysis reinforced this pattern, with participants possessing a family history of PCOD exhibiting higher rates of hypothyroidism, hyperprolactinemia, and elevated ALT compared to those without such a history. These findings corroborate prior studies emphasizing that familial factors can modulate both the endocrine and metabolic phenotypes of PCOS.^{35, 36} Overall, this study aligns with a growing body of evidence indicating that PCOS is not only influenced by individual lifestyle factors and BMI but also significantly shaped by familial and genetic predispositions, which contribute to the heterogeneity in biochemical profiles. Variations in prevalence rates of thyroid dysfunction and hyperprolactinemia compared to other cohorts may reflect differences in ethnicity, sample size, or diagnostic thresholds, underscoring the need for population-specific investigations. Moreover, the observed correlations between family history and biochemical markers suggest that integrated screening of genetic, hormonal, and metabolic parameters could provide a more comprehensive risk assessment for women at higher familial risk.

Limitations of the study

This study had several limitations. The relatively small sample size ($n=50$) may limit statistical power and the ability to detect significant associations. Being cross-sectional, causal relationships between family history and biochemical abnormalities could not be established.

CONCLUSION

The study reveals that family history, especially PCOD, strongly influences biochemical and metabolic profiles in women with PCOS. Positive family histories were linked to higher hypothyroidism, hyperprolactinemia, and elevated ALT. These findings emphasize genetic-metabolic interplay, highlighting the need for family history assessments and targeted monitoring to improve early detection and management.

Recommendations

- Routine assessment of family history, especially first-degree relatives with PCOD.
- Early metabolic and hormonal screening for timely detection.
- Lifestyle interventions and patient education to promote prevention and awareness.

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