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

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ABSTRACT

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 largely physically inactive (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.

INRODUCTION

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 ± 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/m²)	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
	>2.0-High	12	24.00
Serum Prolactin	>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 ± SD (U/L)	Elevated ALT n (%)	p-value
Euthyroid (n=22)	32.1 ± 8.7	5 (22.7)	Reference Group
Hypothyroid (n=26)	42.5 ± 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.

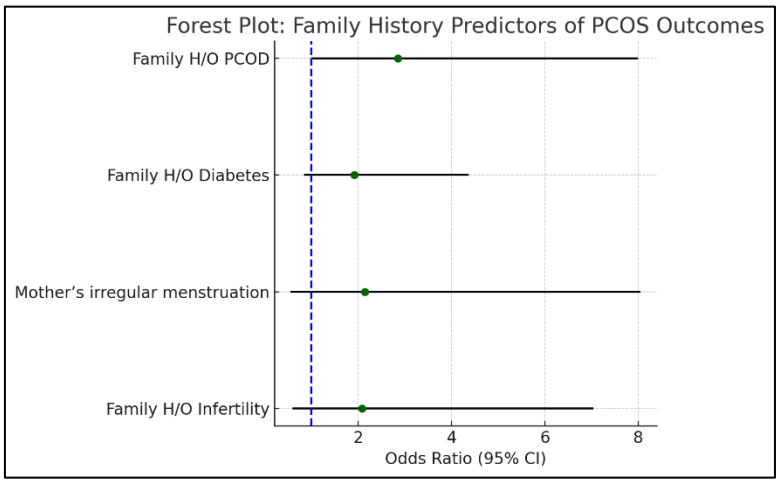

Figure 1: Forest Plot Showing the Impact of Family History Predictors on PCOS Outcomes.

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.

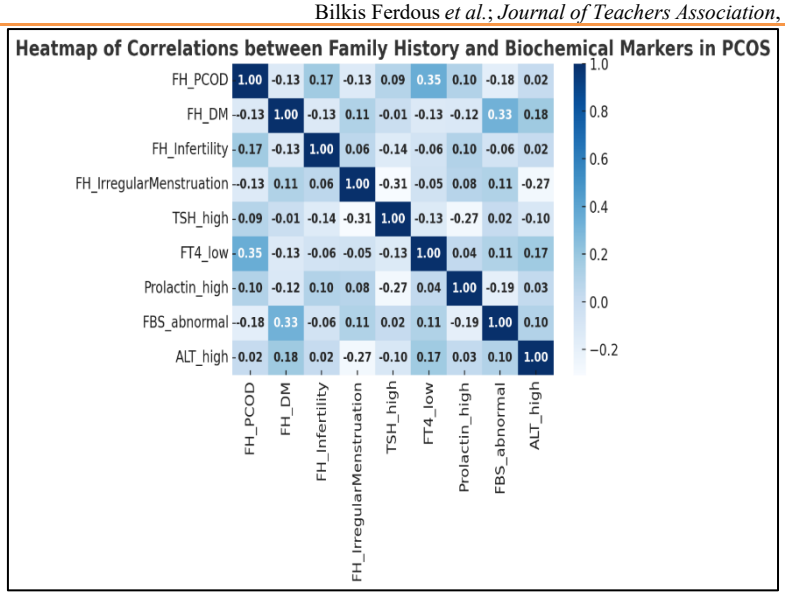

Figure 2: Heatmap of Correlations Between Family History and Biochemical Markers in PCOS.

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.

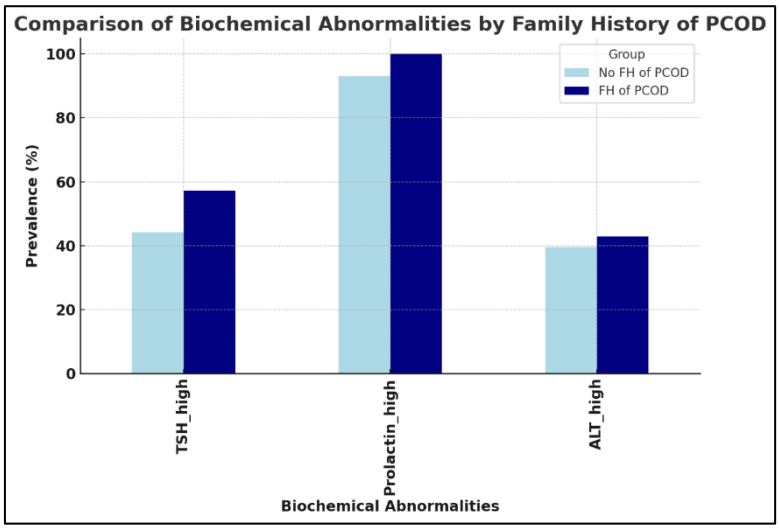

Figure 3: Comparison of Biochemical Abnormalities by Family History of PCOD

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 demonstrates that family history, particularly a direct history of PCOD, significantly influences the biochemical and metabolic profiles of women with PCOS. Participants with positive family histories exhibited higher rates of hypothyroidism, hyperprolactinemia, and elevated ALT, indicating a combined genetic and metabolic predisposition. The findings highlight the strong interplay between familial factors and endocrine dysfunctions, reinforcing the importance of incorporating family history assessments alongside biochemical screening. These insights enhance the understanding of PCOS heterogeneity and suggest that targeted monitoring of at-risk individuals may facilitate early identification of metabolic and hormonal abnormalities.

Recommendation

This study highlights the need to routinely assess family history in women at risk of PCOD, as a positive familial background, particularly first-degree relatives with PCOD, is strongly associated with biochemical abnormalities. Early metabolic and hormonal screening, including thyroid

function, prolactin, glucose tolerance, and liver enzymes, should be considered for timely detection and management. Lifestyle interventions focusing on weight reduction and physical activity remain essential given the high prevalence of obesity and inactivity. Patient education and counselling can improve awareness of the heritable nature of PCOS and encourage preventive health practices. Larger longitudinal studies are recommended to clarify causal links between family history, biochemical markers, and disease progression, and to guide region-specific management strategies.

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REFERENCES

1. Meng Y, Zhao T, Zhang R, Zhu X, Ma C, Shi Q. Global burden of polycystic ovary syndrome among women of childbearing age, 1990–2021: a systematic analysis using the global burden of disease study 2021. Frontiers in Public Health. 2025 Mar 26;13:1514250.
2. Teede HJ, Misso ML, Costello MF, Dokras A, Laven J, Moran L, Piltonen T, Norman RJ. Recommendations from the international evidence-based guideline for the assessment and management of polycystic ovary syndrome. Human reproduction. 2018 Sep 1;33(9):1602-18.
3. Teede HJ, Tay CT, Laven JJ, Dokras A, Moran LJ, Piltonen TT, Costello MF, Boivin J, Redman LM, Boyle JA, Norman RJ. Recommendations from the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome. European journal of endocrinology. 2023 Aug;189(2):G43-64.
4. Ganie MA, Chowdhury S, Malhotra N, Sahay R, Bhattacharya PK, Agrawal S, Jabbar PK, Suri V, Rozati R, Sreenivas V, Baba MS. Prevalence, phenotypes, and comorbidities of polycystic ovary syndrome among Indian women. JAMA Network Open. 2024 Oct 1;7(10):e2440583-.
5. Satapathy P, Vadia N, Menon SV, Chennakesavulu K, Panigrahi R, Bushi G, Singh M, Sah S, Turkar A, Rao SG, Shabil M. Prevalence of Polycystic Ovarian Syndrome in South Asian Countries and 21 Global Regions with Sociodemographic Index: A Systematic Analysis of the Global Burden of Disease (1990–2021). Journal of Gynecology Obstetrics and Human Reproduction. 2025 Aug 27:103019.
6. Kamrul-Hasan AB, Aalpona FT, Mustari M, Selim S. Prevalence and characteristics of women with polycystic ovary syndrome in Bangladesh–A narrative review. Bangladesh Journal of Endocrinology and Metabolism. 2023 Jan 1;2(1):20-8.
7. Sharma NK, Zahan A, Islam M, Bhowmik KR. Prevalence of polycystic ovarian syndrome in women of reproductive age in Mymensingh region of Bangladesh–a sonographic evaluation. Community Based Medical Journal. 2025 Jan 30;14(1):89-95.
8. Welt CK. Genetics of polycystic ovary syndrome: what is new?. Endocrinology and Metabolism Clinics. 2021 Mar 1;50(1):71-82.
9. Ghosh P, Monni AF, Islam MS, Dutta M, Tabassum M, Afsana F, Amin MF, Pathan MF. Study of Serum Calcium, Magnesium, and Fasting Insulin Level in Patients with Polycystic Ovarian Syndrome. Bangladesh Journal of Endocrinology and Metabolism. 2025 May 1;4(2):85-90.
10. Inan C, Karadag C. Correlation between ovarian morphology and biochemical and hormonal parameters in polycystic ovary syndrome. Pakistan Journal of Medical Sciences. 2016 May;32(3):742.
11. Rotterdam ESHRE/ASRM-Sponsored PCOS consensus workshop group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). Hum Reprod. 2004 Jan;19(1):41–7.
12. Azziz R, Carmina E, Chen Z, Dunaif A, Laven JS, Legro RS, Lizneva D, Natterson-Horowtiz B, Teede HJ, Yildiz BO. Polycystic ovary syndrome. Nature reviews Disease primers. 2016 Aug 11;2(1):1-8.
13. Teede HJ, Misso ML, Costello MF, Dokras A, Laven J, Moran L, Piltonen T, Norman RJ. Recommendations from the international evidence-based guideline for the assessment and management of polycystic ovary syndrome. Human reproduction. 2018 Sep 1;33(9):1602-18.
14. Lim SS, Norman RJ, Davies MJ, Moran LJ. The effect of obesity on polycystic ovary syndrome: a systematic review and meta-analysis. Obesity Reviews. 2013 Feb;14(2):95-109.
15. Moran LJ, Misso ML, Wild RA, Norman RJ. Impaired glucose tolerance, type 2 diabetes and metabolic syndrome in polycystic ovary syndrome: a systematic review and meta-analysis. Human reproduction update. 2010 Jul 1;16(4):347-63.
16. Cowan S, Lim S, Alycia C, Pirotta S, Thomson R, Gibson-Helm M, Blackmore R, Naderpoor N, Bennett C, Ee C, Rao V. Lifestyle management in polycystic ovary syndrome–beyond diet and physical activity. BMC endocrine disorders. 2023 Jan 16;23(1):14.
17. Barber TM, Franks S. Genetic and environmental factors in the etiology of polycystic ovary syndrome. InThe Ovary 2019 Jan 1 (pp. 437-459). Academic Press.
18. Vink JM, Sadrzadeh S, Lambalk CB, Boomsma DI. Heritability of polycystic ovary syndrome in a Dutch twin-family study. The Journal of Clinical Endocrinology & Metabolism. 2006 Jun 1;91(6):2100-4.
19. Shi Y, Zhao H, Shi Y, Cao Y, Yang D, Li Z, Zhang B, Liang X, Li T, Chen J, Shen J. Genome-wide association study identifies eight new risk loci for polycystic ovary syndrome. Nature genetics. 2012 Sep;44(9):1020-5.
20. Liu H, Zhao H, Chen ZJ. Genome-wide association studies for polycystic ovary syndrome. InSeminars in reproductive medicine 2016 Jul (Vol. 34, No. 04, pp. 224-

229). Thieme Medical Publishers.
21. Bruni V, Capozzi A, Lello S. The role of genetics, epigenetics and lifestyle in polycystic ovary syndrome development: the state of the art. Reproductive Sciences. 2022 Mar;29(3):668-79.
22. Stener-Victorin E, Teede H, Norman RJ, Legro R, Goodarzi MO, Dokras A, Laven J, Hoeger K, Piltonen TT. Polycystic ovary syndrome. Nature Reviews Disease Primers. 2024 Apr 18;10(1):27.
23. Pervin HH, Kazal RK, Rahman AS, Pervin T, Fatema K, Chowdhury SA, Mamun-Ar-Rashid SK, Mahjabeen S. Thyroid disorders in women with polycystic ovary syndrome. Journal of Biosciences and Medicines. 2020 Apr 24;8(04):128.
24. Bahreiny SS, Ahangarpour A, Amraei M, Mansouri Z, Pirsadeghi A, Kazemzadeh R, Javidan M, Karamali N, Bastani MN, Dabbagh MR. Autoimmune thyroid disorders and polycystic ovary syndrome: Tracing links through systematic review and meta-analysis. Journal of reproductive immunology. 2024 Jun 1;163:104215.
25. Dai F, Wu Z, Qin S, Wu F, Wang M, Liu X, Liu F. The Influence of Subclinical Hypothyroidism on Endocrine and Metabolic Characteristics in Patients with Polycystic Ovary Syndrome. International Journal of Women's Health. 2025 Dec 31:1019-26.
26. Davoudi Z, Araghi F, Vahedi M, Mokhtari N, Gheisari M. Prolactin Level in Polycystic Ovary Syndrome (PCOS): An approach to the diagnosis and management. Acta Bio Medica: Atenei Parmensis. 2021 Nov 3;92(5):e2021291.
27. Chaudhary, K., Ghosh, S., Mitra, P., & Pradhan, A. (2025). Prevalence of hyperprolactinemia in polycystic ovary syndrome and its clinical correlates. *International Journal of Reproduction, Contraception, Obstetrics and Gynecology*, *14*(9), 3034–3039. https://doi.org/10.18203/2320-1770.ijrcog20252737
28. Hong SH, Sung YA, Hong YS, Song DK, Jung H, Jeong K, Chung H, Lee H. Non-alcoholic fatty liver disease is associated with hyperandrogenism in women with polycystic ovary syndrome. Scientific Reports. 2023 Aug 17;13(1):13397.
29. Khan RS, Bril F, Cusi K, Newsome PN. Modulation of insulin resistance in nonalcoholic fatty liver disease. Hepatology. 2019 Aug;70(2):711-24.
30. Anagnostis P, Tarlatzis BC, Kauffman RP. Polycystic ovarian syndrome (PCOS): Long-term metabolic consequences. Metabolism. 2018 Sep 1;86:33-43.
31. Chen MJ, Ho HN. Hepatic manifestations of women with polycystic ovary syndrome. Best practice & research Clinical obstetrics & gynaecology. 2016 Nov 1;37:119-28.
32. Fan H, Ren Q, Sheng Z, Deng G, Li L. The role of the thyroid in polycystic ovary syndrome. Frontiers in endocrinology. 2023 Oct 5;14:1242050.
33. Su P, Chen C, Sun Y. Physiopathology of polycystic ovary syndrome in endocrinology, metabolism and inflammation. Journal of ovarian research. 2025 Feb 20;18(1):34.
34. Goodarzi MO, Dumesic DA, Chazenbalk G, Azziz R. Polycystic ovary syndrome. Etiology, pathogenesis and diagnosis. 2016;2011:7.
35. Wang Y, Gao H, Di W, Gu Z. Endocrinological and metabolic characteristics in patients who are non-obese and have polycystic ovary syndrome and different types of a family history of type 2 diabetes mellitus. Journal of International Medical Research. 2021 May;49(5):03000605211016672.
36. Khan MJ, Ullah A, Basit S. Genetic basis of polycystic ovary syndrome (PCOS): current perspectives. The application of clinical genetics. 2019 Dec 24:249-60.

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