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RESEARCH ARTICLE
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"FAMILY HISTORY OF DIABETES AS A SIGNIFICANT RISK FACTOR FOR POLYCYSTIC OVARY SYNDROME (PCOS)"

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common endocrine disease with major metabolic consequences. Having type 2 diabetes clustered among families might imply that there is shared and inherited insulin resistance that may make the individual susceptible to PCOS and worsen its metabolic phenotype.

Objective: To determine whether the first-degree family history of type 2 diabetes is a prominent risk factor of PCOS, among reproductive-aged women who attend Primary health care.

Methods: The cross-sectional study was conducted at Primary health care setting in the duration from January, 2025 to December, 2025. The age of enrolled women included that of 18-40. PCOS was diagnosed by using Rotterdam criteria after the secondary causes were excluded. The first-degree relatives were taken to be of type 2 diabetes because of the family history of diabetes. Anthropometric measurements, clinical measurements, and the measurement of fasting glucose were done. Associations were tested using chi-square and t-tests. Binary logistic regression delivered adjusted odds ratios (AOR) that confounded on the age as well as the BMI.

Results: 104 (32.5) participants among 320 participants in the study completed the requirements of PCOS. Hundred and eighty (36.9 percent) reported having a family history of diabetes in the 1 st degree. It was also established that the prevalence of PCOS was greater in women with a positive family history (44.1) than in those with a negative one (25.7) ($p=0.001$). Multivariate analysis also showed a positive correlation between family history of diabetes and PCOS (AOR 2.05; 95 percent confidence interval 1.26-3.33; $p=0.004$). PCOS was additionally also correlated with BMI independently (AOR 1.09 /kg/m2; $p<0.001$), but not with age.

Conclusion: The PCOS risk factor of diabetes that is independent of others is the first-degree family history of diabetes. Conducting routine screening of recent family history of diabetes can improve the process of early diagnosis and metabolic screening of the high-risk women.

Keywords: Polycystic ovary syndrome; family history; type 2 diabetes mellitus; insulin resistance; reproductive-age women.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is an endocrine disorder that is common in women aged between reproductive age with hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, having a broad heterogeneity in its manifestation. In addition to reproductive manifestations, PCOS is now becoming perceived as a systemic metabolic disorder in which insulin resistance, dysglycemia,

and cardiometabolic susceptibility are concentrated in the early life pathway and maintained throughout the life cycle. The primary limitation is the fact that metabolic risk of PCOS does not limit itself to obese women, which implies that genetic predisposition and familial-related metabolic characteristics could have a significant impact on the manifestation of the disease. In non-obese PCOS, endocrinological and metabolic patterns have been described based on the existence of a family history of type 2 diabetes, which leads to the assumption that the family diabetes history is not just a comorbidity indicator but rather a biologically significant risk factor of metabolic dysregulation in PCOS (1). Women with PCOS are highly exposed to hypertensive complications in pregnancy and the profiles of risk factors that were identified suggest that metabolic and vascular pathways overlap the syndrome in clinically significant manners (2).

This correlates with the perception that PCOS represents an inherent risk condition leading to diabetes which is increased by adiposity but not entirely accounted by it and, therefore, the relative importance of the familial risk of diabetes is of special significance in situations where obesity is absent or mediocre (3). Gestational and later-life dysglycemia are high-stakes outcomes, where the PCOS-diabetes relationship becomes clinically evident, and the literature is increasingly adopting the view that PCOS is a condition that can reveal the existence of latent diabetogenic risk in genetically susceptible families. Synthetic synthesis suggests that gestational diabetes mellitus (GDM) is more prevalent in women who are PCOS and the involved risk factors are not limited to body mass index, but also to metabolic history and vulnerabilities, which support the justification of analyzing familial diabetes patterns as upstream determinants (4). PCOS-related predictive modeling studies have also focused on predicting risks among high-risk women, suggesting that it is possible to identify high-risk women and their clinical prognostication can be enhanced in combination with reproductive and anthropometric factors (5).

Importantly, there is specific evidence in Primary health care setting that family history of diabetes is a significant risk factor in development of PCOS, which supports the feasibility of the idea that in high and increasing diabetes populations, familial clustering of dysglycemia may play a significant role in the development and severity of phenotype of PCOS (6). The connection between PCOS and type 2 diabetes is not limited to pregnancy and it has long-term vascular outcome implications which increases the significance of early detection of familial metabolic risk even further. Massive analyses and reviews have associated PCOS to higher risk of type 2 diabetes and substantial cardiometabolic consequences as coronary heart disease and stroke, thus suggesting that PCOS can also serve as a sentinel condition prior to cardiometabolic disease, especially when metabolic danger is already established within families (7). Speaking specifically of the risk factors of GDM in PCOS, there is a clinical literature that suggests a need to pay attention to both modifiable and non-modifiable risk factors, which suggests the possibility that family history may be a feasible inexpensive risk factor in resource-intensive triage settings (8).

Genetic susceptibility data add to this argument: the history of diabetic-like family history, combined with menstrual irregularity history has been demonstrated to increase the susceptibility to PCOS in individuals with a history of inherited glycemic risk in the form of diabetogenic variants of TCF7L2, such as the rs7903146, implicating a mechanistic relationship between inherited glycemic risk history and the development of PCOS, and not merely an epidemiologic correlation (9). The lived epidemiology of the PCOS also has a family history, which interacts with mental health, nutrition, and cardiometabolic risk that have broader outcome effects and healthcare utilization. The family history has been used as a significant correlate in questionnaire-based assessment reporting the prevalence of PCOS and its relationship with psychosocial variables, such as depressive symptoms, which is significant as the symptom burden and seeking healthcare can impact the diagnosis of PCOS (10). In terms of cardiovascular prevention, PCOS has been contended to be a risk-enhancing factor to cardiovascular disease, and that the classic risk algorithms might not be sufficiently sensitive to risk in these women and that family history of diabetes may also increase lifetime cardiometabolic risk exposure (11).

Similar conclusions on the interdependence of cardiometabolic disease pathways in PCOS are drawn on the basis of narrative syntheses underscoring that the history of familial diabetes is plausibly a

carrier of inherited and shared-environment risks converging at pathways and potentially pre-disposing to the development or exacerbation of PCOS (12). The reproductive decision making and clinical management of PCOS demand a broader perspective that encompasses familial metabolic risk especially with the prevalence of pregnancy related complications and the necessity of a tailored contraceptive and metabolic counseling. A systematic review of the GDM as a PCOS complication supports the idea that this syndrome is not a reproductive disease, but a condition with a predictable metabolic impact, and risk assessment should start early, preferably before conception, and family history may serve as a reliable risk factor in this scenario (13). Metabolic heterogeneity should also be incorporated into contraceptive counseling in PCOS since hormonal options can affect weight, glucose tolerance and lipid levels hence, a history of diabetes in the family can reasonably alter counseling to methods and monitoring strategies that cause the fewest metabolic damage and achieve reproductive objectives (14).

Recent cohort data regarding risk factors involved in incident PCOS diagnosis points to the fact that PCOS is brought about by a cluster of determinants, but not necessarily by one, and therefore it would be scientifically sound to test family history of diabetes as an important independent predictor, at least in combination with other demographic, reproductive, and metabolic covariates (15). Collectively, the literature promotes an etiologic model of PCOS as being the overlap of female reproductive endocrine disruption and hereditary or familial metabolic susceptibility with a family history of diabetes being a clinically available surrogate of that susceptibility. Case-control data reveal several risk factors of PCOS in women who are of reproductive age and support the idea that risk patterns within the population can be different based on the population context and that familial history of diabetes is likely to be a strong predictor of risk in different contexts but are not likely to have similar risk architecture (16).

Genetic research also offers greater detail, in showing findings that ovary-independent effects of genetic risk factors of PCOS phenotype do exist, suggesting that systemic metabolic-endocrine pathways, which might exist between sexes, may be associated with PCOS liability; thus supporting the possibility that familial diabetes risk and shared genetic architecture may be contributors of PCOS development in the female long before metabolic disease has manifested in that female herself (17). The most recent syntheses of PCOS and type 2 diabetes also further solidarize the opinion that the relationship between the two conditions is reciprocal in clinical importance: that PCOS predicts the risk of dysglycemia, and that the prevalence of diabetes related-family and genetic predisposition potentially drives PCOS phenotype and course in the first place, as family history of diabetes is a scientifically viable and clinically actable factor to explore as an important risk predictor of PCOS in the hospital population (18).

Objective: To ascertain the significance of a family history of diabetes with PCOS in reproductive-age women who visit Primary health care setting and to estimate the strength of the association between family history of diabetes and PCOS after adjusting the effects of important clinical variables.

MATERIALS AND METHODS

Study Design: Cross sectional Study.
Study Setting: Primary health care centers in South west England.
Duration of Study: 1 year (January, 2025 to December, 2025).
Sample Size: A total of 320 participants, calculated using a 95% confidence level, 5% margin of error, and expected PCOS prevalence in reproductive-age women, with allowance for non-response.
Inclusion Criteria: The eligibility criteria were women aged 18 years to 40 years presenting to gynecology OPD. The inclusion criteria were informed consent and clinical assessment and ultrasound to determine the PCOS status.

Exclusion Criteria: Those women who had a known thyroid disease, hyperprolactinemia, congenital adrenal hyperplasia, Cushing syndrome, androgen secreting tumors, were excluded as well as those

who are pregnant and those who are taking drugs that influence hormones or glucose metabolism (within the last three months).

Methods

The diagnosis of PCOS was based on Rotterdam criteria (any two out of: oligo/anovulation, clinical/biochemical hyperandrogenism, polycystic ovarian morphology on ultrasound) since secondary causes were excluded. A detailed questionnaire was used to tell age, marital status, BMI, menstrual history, clinical evidence of hyperandrogenism, and obstetric history. Type 2 diabetes among the first-degree relatives (mother, father, or siblings) was considered as family history of diabetes. Standard procedures were used in anthropometry. Blood samples were fasted and checked to measure glucose and (where possible) insulin and lipid profile, ultrasound was conducted under the supervision of a trained sonologist. Data were evaluated with help of SPSS. The dependent variable was PCOS status and the primary exposure was the family history of diabetes. The chi-square and independent t-tests were used to compare categoric and continuous variables, respectively. Binary logistic regression was used to estimate adjusted odds ratios at 95 percent confidence interval age and BMI controlling. The p-value of less than 0.05 was employed as a statistically significant value.

Results

There was the enrolment of 320 women. The average age was 26.90 years with a standard deviation of 5.4 years and the average BMI was 27.30kg/m 2with a standard deviation of 4.8. A total of 104 subjects were eligible using Rotterdam criteria to PCOS providing a prevalence of 32.5. The number of women with type 2 diabetes mellitus that had a first-degree family history of the disease was 118 (36.9%). Women who were PCOS had mean BMI and a larger percentage of menstrual irregularity and clinical hyperandrogenism than non-PCOS women.

Table 1 shows a summary of baseline characteristics. Cases of PCOS were more apt to be overweight/obese and to report of interference of menstrual cycle. Clinical hyperandrogenism (hirsutism/acnes) too was more common in the PCOS cases as per the diagnostic criteria. The participants with PCOS also had a minor higher value in fasting glucose, but the majority of the values were in the non-diabetic range, indicating that participants were prone to metabolic issues early on, as opposed to being already diagnosed with diabetes.

Table 1. Baseline characteristics of participants (n=320)

Variable	PCOS (n=104)	Non-PCOS (n=216)	p-value
Age (years), mean ± SD	26.4 ± 5.2	27.1 ± 5.5	0.24
BMI (kg/m²), mean ± SD	29.0 ± 4.9	26.5 ± 4.5	<0.001
BMI category, n (%)			0.002
• Normal (<25)	18 (17.3)	72 (33.3)	
• Overweight (25–29.9)	46 (44.2)	92 (42.6)	
• Obese (≥30)	40 (38.5)	52 (24.1)	
Menstrual irregularity, n (%)	79 (76.0)	54 (25.0)	<0.001
Clinical hyperandrogenism, n (%)	62 (59.6)	41 (19.0)	<0.001
Fasting glucose (mg/dL), mean ± SD	94.8 ± 10.6	90.9 ± 9.7	0.001

PCOS was strongly related with family history of diabetes. The positive family history of PCOS was found in 52/118 (44.1) of women as compared to 52/202 (25.7) of women with no family history. The crude odds of PCOS were found to be two times higher among women who reported diabetes among their first-degree relatives.

Table 2. Association of family history of diabetes with PCOS (n=320)

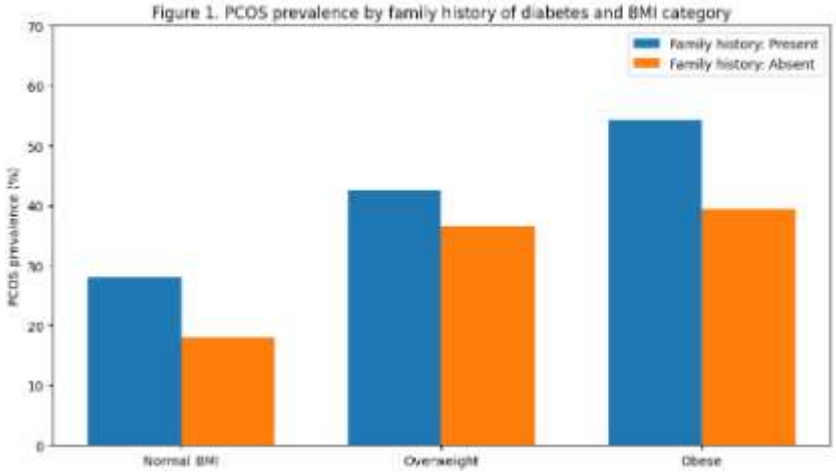
Family history of diabetes	PCOS n (%)	Non-PCOS n (%)	Total	p-value
Present	52 (44.1)	66 (55.9)	118	0.001
Absent	52 (25.7)	150 (74.3)	202	

Family history of diabetes was also independently related to PCOS in the case of multivariate analysis but the ages and BMI were controlled. BMI was also found to positively correlate with PCOS with no statistically significant difference in the adjusted model with age. The results suggest that family history of diabetes plays a role over existing adiposity, which is in favor of a familial-metabolic susceptibility pathway.

Table 3. Binary logistic regression for predictors of PCOS (n=320)

Predictor	Adjusted OR	95% CI	p-value
Family history of diabetes (present vs absent)	2.05	1.26–3.33	0.004
BMI (per 1 kg/m² increase)	1.09	1.04–1.14	<0.001
Age (per 1 year increase)	0.98	0.94–1.02	0.33

Figure 1. PCOS prevalence increases with BMI, and within each BMI category remains higher in women with a family history of diabetes.



Discussion

We have discovered that a PCOS that women who visit the gynecology outpatient department had a significant association with family history of type 2 diabetes (first degree), despite age and BMI factors. This is congruent with findings that the family history of diabetes could determine the endocrine-metabolic phenotype of PCOS even in non-obese individuals, indicating a familial metabolic vulnerability that cannot be adequately explained by anthropometry per se (1). This relationship would be clinically significant since PCOS is often accompanied by metabolic upheavals that convert to poor obstetric consequences. We did not study it as pregnancy-specific phenomenon, but the more encompassing PCOS phenotype that we found (greater BMI, irregular menstruation, hyperandrogenic appearance) overlaps with the profiles associated with effects of pregnancy complications including gestational hypertension, suggesting that family-based metabolic risk can be co-morbid with vascular susceptibility (2). One important meaning is that PCOS could be intrinsic diabetogenic condition where insulin resistance and stress on 3 pancreatic cells exists early and obesity is only an amplifier and not a causative factor. The independent effects of family history of diabetes and BMI were observed in our regression model, which is agreeable to the fact that in PCOS, the metabolic risk at baseline remains independent of statistical control by adiposity (3).

It is the family history that is especially significant when we are talking about the continuum between preclinical dysglycemia and overt diabetes in PCOS. There are systematic data that suggest increased incidences of gestational diabetes in PCOS and that a variety of contributory risks exist independent of weight, and thus our conclusion that a family history of diabetes may be an upstream factor that predisposes an individual to metabolic decompensation under physiologic stress (4). Regarding risk-stratification, we find evidence that family history is a practical screening variable to be used in normal gynecology practice. Prognostic models of gestational diabetes in pregnant women with PCOS focus on the inclusion of the easily accessible clinical data in formalized risk systems, family history of diabetes is stable, cost-effective. The fact that this relation has been reproduced in different settings supports the possibility that there is a joint impact of shared genes and shared household exposures (dietary patterns, physical inactivity, socioeconomic constraints) to create the risk of PCOS (6).

This association is not limited to reproductive symptoms since PCOS has a future cardiometabolic outcome, such as type 2 diabetes and macrovascular disease. In the event of family history of diabetes signifying an elevated baseline metabolic pattern, then PCOS in these women may suggest a subgroup at especially great lifetime risk, which ought to be prevented earlier (7). The fact that fasting glucose was marginally elevated in the PCOS group is consistent with clinical practice that points at the need to give substantial attention to metabolic risk factors in PCOS, such as those that are antecedents of diabetes. Practically, this is applicable by placing priority in fasting glucose, HbA1c, and lifestyle counseling as offered by family history of diabetes, particularly in the hectic outpatient clinics (8).

Genetic evidence is growing in support of the mechanistic association between family history of diabetes and PCOS. Some studies indicate that a familial history of diabetes may amplify the predisposition of individuals that carry diabetogenic variants (such as TCF7L2) to PCOS, which indicates that the two phenomena are biologically coupled, not merely coincidentally overlapping (9).

Psychosocial and behavioral factors also interact with family history in the diagnosis and outcomes. The work conducted through questionnaires has linked PCOS to depression, nutrition related problems and family history trends, and suggests that women having family history metabolic disease, can both have increased symptom burden and increased healthcare utilization which can influence detection, counseling uptake and long-term compliance (10). PCOS is becoming a condition that is a risk-enhancing condition according to cardiovascular prevention frameworks. In this respect, a positive family history of diabetes can be another dimension of risk enrichment that justifies a more vigorous method of screening blood pressure abnormalities, dyslipidemia, and glycemic abnormalities and insists on early intervention in lifestyle as its primary prevention (11). Convergent mechanisms that have been proposed in narrative syntheses of cardiometabolic disease in PCOS are insulin resistance, low-grade inflammation, dyslipidemia, and endothelial dysfunction. The independent effect of family history that we have observed is also in line with the premise that inherited predisposition and common environmental exposures accelerate these pathways, thus facilitating the expression of PCOS and metabolic degeneration (12).

Even though the results of pregnancy were not a part of our dataset, the correlation that we observed can be applied to the setting of the antenatal risk plans as women with PCOS are more prone to gestational diabetes in systematic reviews. Presence of a family history of diabetes can also contribute to that risk, and in this case, preconception counseling is supposed to involve a particular discussion of familial metabolic disease (13). The implications of reproductive management are spread to the contraceptive counseling. Modern advice emphasizes personal choice of contraception in PCOS, because there is heterogeneity in metabolic risk. Clinicians in women with a diabetic family history can be rightful in focusing on options and follow-up strategies that reduce adverse metabolic changes and guarantee a periodic check of weight and glycemic indexes (14). Our findings are also in line with cohort data to establish several factors of incident PCOS diagnosis. It means that family history of diabetes can be viewed as a component of a multi-factor risk architecture, which interacts with BMI, symptom patterns, and access to healthcare as opposed to a secondary background factor (15).

The cross-population data also in favor of the familial and metabolic factors in PCOS risk is evident. The case-control results suggest that the determinants of PCOS are in the form of a bundle with different weights in different settings. Family history of diabetes could also be a strong predictor in

this kind of setting since it incorporates inherited and shared lifestyle risks (16). The results of genetic studies which mention ovary-independent effects of PCOS risk variants offer another interpretive dimension: systemic pathways that affect metabolism and endocrine regulation might play a role in PCOS liability without ovarian-specific effects. This enhances the possibility that, a family history of diabetes represents common genetic design that elevates PCOS vulnerability (17). Lastly, recent advanced reviews of the PCOS and type 2 diabetes focus on the fact that the linkage is clinically interventional since early detection of metabolic vulnerability can change long-term patterns. We have found that the first-degree family history of diabetes is an easy yet useful variable that should be prioritized in the initial screening and preventive counseling of women with PCOS as it appears (18). On the whole, the research indicates that family history of diabetes should be included in the normal PCOS risk evaluation during the first contact of the gynecology outpatient care. The chief weaknesses are cross-sectional nature, which limits causal inference, and the use of self-reported family history, which may result in misclassification; nevertheless, the observed association was robust after adjustment and agrees with convergent international evidence within metabolic, genetic, and clinical fields.

Conclusion

This paper shows that a history of type 2 diabetes in the first degree family is quite strongly connected to PCOS development among gynecological outpatient department women of reproductive age. Women with a history of diabetes in close family members had near a twofold probability of having PCOS even after adjusting age and BMI, which shows that familial metabolic proneness adds on its own to present-day obesity. These results can be used to back the idea that PCOS is not only a reproductive endocrine illness, but there is also inherited and shared-environment metabolic risk. Family history of diabetes is a relatively inexpensive, but straightforward, clinical measure, which may enhance risk prioritization at an early stage and inform the time-prompted screening of dysglycemia, weight control, and cardiometabolic risk factors in women with suspected or confirmed PCOS. Since the study is cross-sectional, longitudinal research would be further suggested to prove the time-related associations and to investigate whether family-based prevention interventions in high-risk families can decrease the severity of PCOS and the risk of developing diabetes.

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