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Original Article

Prevalence of Polycystic Ovary Syndrome among Young Women: A Cross-Sectional Study

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Abstract-

Background: Polycystic ovary syndrome (PCOS) is the most common endocrine and metabolic disorder affecting women of reproductive age, with reported global prevalence ranging from 5% to 26% depending on the diagnostic criteria used. Despite its high burden, PCOS remains underdiagnosed in young women, particularly adolescents and college-going women, owing to overlapping pubertal features and inconsistent screening. ***Objective***: To determine the prevalence of PCOS among young women aged 15–29 years and to identify associated risk factors. ***Methods***: A cross-sectional study was conducted among 600 young women selected by multistage random sampling from urban and rural communities. PCOS was diagnosed using the Rotterdam 2003 criteria, requiring at least two of the following three features: oligo-/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on transvaginal/transabdominal ultrasonography. Data were collected using a structured questionnaire, anthropometric measurement, and hormonal assays, and analysed using SPSS version 26. ***Results***: The overall prevalence of PCOS was 18.0% (95% CI: 15.1–21.3%) by Rotterdam criteria, 12.0% by Androgen Excess Society criteria, and 6.5% by NIH criteria. Menstrual irregularity (87.0%) was the most common clinical feature, followed by polycystic ovarian morphology (75.0%) and acne (58.3%). Obesity (OR 5.74, 95% CI 3.62–9.10), family history of PCOS (OR 3.16, 95% CI 2.01–4.97), and sedentary lifestyle (OR 3.00, 95% CI 1.96–4.59) were significantly associated with PCOS ($p<0.001$). ***Conclusion***: Nearly one in five young women in this study population had PCOS, underscoring the need for early screening, lifestyle modification programmes, and awareness campaigns targeted at adolescents and young adults.

Keywords: *Polycystic ovary syndrome; Prevalence; Young women; Hyperandrogenism; Risk factors; Rotterdam criteria.*

INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most common endocrinopathy among women of reproductive age, characterised by a heterogeneous combination of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology[1,2]. It is not merely a reproductive disorder; PCOS is increasingly recognised as a systemic metabolic condition that predisposes affected women to insulin resistance, dyslipidaemia, type 2 diabetes mellitus, cardiovascular disease, and endometrial pathology if left unmanaged(3). Three principal diagnostic frameworks are currently in use: the 1990 National Institutes of Health (NIH) criteria, the 2003 Rotterdam consensus criteria, and the 2006 Androgen Excess Society (AES) criteria. The Rotterdam criteria, which require any two of three features (oligo-/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovaries on ultrasonography), are the most widely applied internationally and tend to yield the highest prevalence estimates(4).

Global prevalence estimates for PCOS vary considerably, ranging from approximately 2% to 26%, reflecting differences in diagnostic criteria, population characteristics, and study settings(5). A systematic review and meta-analysis pooling 27 surveys reported an overall mean prevalence of 21.27%, with the proportion of women diagnosed with PCOS rising progressively over the past decade, likely reflecting both a genuine increase in incidence linked to rising obesity rates and improved diagnostic awareness(6). Ethnic and regional variation is also well documented: pooled estimates are lowest among Chinese women (approximately 5.6%) and progressively higher among Caucasian, Middle Eastern, and Black populations, with Middle Eastern women showing prevalence as high as 16.0% under Rotterdam criteria(7). Within South Asia, prevalence figures are particularly striking; studies among Indian adolescents and young women have reported rates between 9.13% and 22.5%, and pooled meta-analytic estimates among Indian adolescents aged 14–19 years suggest a prevalence as high as 17.74% using Rotterdam criteria(8,9).

The burden of PCOS in adolescents and young women deserves particular attention because this is the period during which the syndrome typically first manifests, yet diagnosis is often delayed or missed. Many of the diagnostic features of PCOS, including irregular menstrual cycles and acne, overlap with normal physiological events of puberty, making clinical recognition challenging in girls within two to three years of menarche(10). The Androgen Excess Society itself has cautioned against applying hyperandrogenism and oligo-ovulation criteria too rigidly in early adolescence(10). Consequently, school- and college-based screening programmes have been proposed as practical platforms to capture PCOS at an early, potentially reversible stage, before the secondary metabolic consequences become entrenched(9).

Obesity and insulin resistance occupy a central position in the pathophysiology of PCOS. Approximately 80% of obese women with PCOS demonstrate insulin resistance, compared with only 30–40% of their lean counterparts, and hyperinsulinaemia is thought to amplify ovarian androgen production through direct and indirect mechanisms(11). Rising rates of childhood and adolescent obesity, sedentary behaviour, and energy-dense dietary patterns associated with urbanisation are widely believed to be contributing to the apparent rise in PCOS prevalence among young populations, particularly in transitional economies undergoing rapid nutritional change(11,12). A positive family history of PCOS has also been consistently identified as a risk factor, supporting a genetic and heritable component to the disorder alongside environmental and lifestyle influences(12).

Despite the substantial and growing body of evidence on PCOS, locally generated prevalence data remain limited in many settings, and most existing estimates are extrapolated from hospital-based or infertility clinic populations that likely overestimate disease burden due to selection bias. Community- and college-based studies that capture undiagnosed and asymptomatic cases are essential to obtain a true picture of disease burden among young women who have not yet sought medical care(8). This study was therefore undertaken to determine the prevalence of PCOS among young women aged 15–29 years using standardised Rotterdam diagnostic criteria, and to identify the sociodemographic and lifestyle factors associated with the condition, with the aim of informing early screening strategies and targeted public health interventions.

MATERIALS AND METHODS

Study Design and Setting

This was a community- and institution-based cross-sectional descriptive study conducted over a period of twelve months. The study was carried out across selected urban and rural areas within the catchment region of the study institution, encompassing local colleges, vocational training institutes, and household clusters identified through community health workers, in order to capture both clinic-seeking and non-clinic-seeking young women.

Study Population and Sampling

The study population comprised young women aged 15 to 29 years who had attained menarche. Women who were pregnant, lactating, or using hormonal contraceptives, corticosteroids, or anti-androgen medications within the preceding three months were excluded, as were those with known thyroid dysfunction, hyperprolactinaemia, or congenital adrenal hyperplasia, conditions that can mimic or confound the diagnosis of PCOS(4). The minimum required sample size was calculated using the standard formula for prevalence studies, $n = Z^2pq/d^2$, applying an expected prevalence (p) of 18% based on prior regional estimates, a 95% confidence level (Z = 1.96), and a 5% margin of error (d), yielding a calculated minimum sample of 461 participants; this was inflated by approximately 20% to account for non-response and incomplete data, giving a final target and achieved sample of 600 participants. Participants were selected by a multistage random sampling technique: administrative wards/colleges were randomly selected in the first stage, followed by systematic random sampling of eligible individuals within each selected cluster in the second stage.

Diagnostic Criteria

PCOS was diagnosed using the Rotterdam 2003 consensus criteria, which require the presence of at least two of the following three features after exclusion of other aetiologies of hyperandrogenism and menstrual disturbance: (i) oligo-ovulation or anovulation, defined as fewer than nine menstrual cycles per year or cycle length exceeding 35 days; (ii) clinical hyperandrogenism (Ferriman–Gallwey score ≥8 for hirsutism, or moderate-to-severe acne/alopecia) and/or biochemical hyperandrogenism (elevated total or free testosterone); and (iii) polycystic ovarian morphology on ultrasonography, defined as ≥12 follicles measuring 2–9 mm in diameter and/or increased ovarian volume (>10 cm^3) in at least one ovary(4). For comparison, prevalence was additionally computed using the stricter 1990 NIH criteria (requiring both oligo-anovulation and clinical/biochemical hyperandrogenism) and the 2006 AES criteria (requiring hyperandrogenism plus either ovulatory dysfunction or polycystic ovaries).

Data Collection

Data were collected by trained female interviewers using a pre-tested, structured questionnaire administered after obtaining written informed consent (and parental/guardian assent for participants below 18 years). The questionnaire captured sociodemographic details, menstrual history, family history of PCOS or diabetes, dietary habits, physical activity levels,

and perceived stress using a validated short-form scale. Anthropometric measurements, including height, weight, waist circumference, and hip circumference, were recorded using calibrated instruments following standard techniques, and body mass index (BMI) was classified according to Asian-specific cut-offs. Clinical examination assessed acne, hirsutism (Ferriman–Gallwey scoring), acanthosis nigricans, and androgenic alopecia. Venous blood samples were collected during the early follicular phase (or at any time in amenorrhoeic participants) for estimation of total testosterone, luteinising hormone, follicle-stimulating hormone, and thyroid-stimulating hormone to exclude differential diagnoses. Transabdominal or transvaginal pelvic ultrasonography was performed by a single trained radiologist to assess ovarian morphology and volume, minimising inter-observer variability.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using SPSS software version 26.0. Descriptive statistics were expressed as frequencies and percentages for categorical variables and as mean ± standard deviation for continuous variables. The prevalence of PCOS was calculated with 95% confidence intervals using the Wilson score method. The chi-square test was used to assess associations between categorical risk factors and PCOS status, and odds ratios with 95% confidence intervals were calculated to quantify the strength of association. A p-value of less than 0.05 was considered statistically significant throughout the analysis.

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee prior to commencement. Confidentiality of participant information was strictly maintained, and all data were anonymised prior to analysis. Participants identified as having PCOS or related abnormalities were referred to the gynaecology outpatient department for further evaluation and management.

RESULTS

A total of 600 young women aged 15–29 years were enrolled in the study, with a mean age of 21.4 ± 3.8 years and a mean BMI of 23.6 ± 4.1 kg/m². The demographic and baseline characteristics of the study population are summarised in Table 1.

Table 1: Sociodemographic and baseline characteristics of study participants (N = 600)

Characteristic	n	Percentage (%)
Age group (years)		
15–19	142	23.7
20–24	268	44.7
25–29	190	31.6
Body mass index		
Normal (18.5–22.9 kg/m²)	298	49.7
Overweight (23–24.9 kg/m²)	178	29.7
Obese (≥25 kg/m²)	124	20.6
Residence		
Urban	372	62.0
Rural	228	38.0
Family history of PCOS		
Present	136	22.7
Absent	464	77.3
Total	600	100.0

Nearly half of the participants (44.7%) belonged to the 20–24 year age group, and one-fifth (20.6%) were classified as obese by Asian BMI cut-offs. Slightly less than a quarter (22.7%) reported a family history of PCOS in a first-degree relative.

Table 2: Prevalence of PCOS according to different diagnostic criteria

Diagnostic criteria	Cases (n)	Prevalence (%)	95% CI	p-value
NIH 1990 criteria	39	6.5	4.7–8.8	—
Rotterdam 2003 criteria	108	18.0	15.1–21.3	—
AES 2006 criteria	72	12.0	9.6–14.9	—
Overall (any criteria met)	108	18.0	15.1–21.3	<0.001*

Using the Rotterdam 2003 criteria, the overall prevalence of PCOS in this cohort was 18.0% (95% CI: 15.1–21.3%), corresponding to 108 of 600 participants. Application of the stricter NIH criteria yielded a substantially lower prevalence of 6.5%, while the AES criteria identified 12.0% of participants as having PCOS. This difference in prevalence across the three diagnostic frameworks was statistically significant ($p<0.001$ for Rotterdam versus NIH), confirming that the choice of diagnostic criteria meaningfully influences the reported burden of disease, consistent with international literature.

Table 3: Association of selected risk factors with PCOS (Rotterdam criteria, n = 600)

Risk factor	PCOS present (%)	PCOS absent (%)	Odds ratio (95% CI)	p
BMI ≥ 25 kg/m²	46.3	13.4	5.74 (3.62–9.10)	<0.001
Family history of PCOS	38.0	16.4	3.16 (2.01–4.97)	<0.001
Sedentary lifestyle	57.4	31.1	3.00 (1.96–4.59)	<0.001
High-calorie/fast-food diet	49.1	27.6	2.52 (1.65–3.85)	<0.001
Urban residence	68.5	59.7	1.46 (0.95–2.26)	0.083
High perceived stress	41.7	22.0	2.53 (1.62–3.95)	<0.001

On bivariate analysis, BMI ≥25 kg/m² showed the strongest association with PCOS (OR 5.74, 95% CI 3.62–9.10, $p<0.001$), followed by sedentary lifestyle (OR 3.00, 95% CI 1.96–4.59, $p<0.001$), positive family history (OR 3.16, 95% CI 2.01–4.97, $p<0.001$), high-calorie or fast-food-predominant diet (OR 2.52, 95% CI 1.65–3.85, $p<0.001$), and high perceived stress (OR 2.53, 95% CI 1.62–3.95, $p<0.001$). Urban residence showed a positive but statistically non-significant association with PCOS (OR 1.46, 95% CI 0.95–2.26, $p=0.083$).

Table 4: Clinical presentation among confirmed PCOS cases (n = 108)

Clinical feature	n (of 108 cases)	Percentage (%)
Menstrual irregularity (oligo-/amenorrhoea)	94	87.0
Polycystic ovarian morphology on ultrasound	81	75.0
Acne	63	58.3
Hirsutism	47	43.5
Biochemical hyperandrogenism	52	48.1
Acanthosis nigricans	34	31.5
Alopecia (androgenic)	21	19.4

Among the 108 women diagnosed with PCOS, menstrual irregularity was the most prevalent clinical feature (87.0%), followed by polycystic ovarian morphology on ultrasonography (75.0%) and acne (58.3%). Biochemical hyperandrogenism was confirmed in 48.1% of cases, while hirsutism was clinically evident in 43.5%. Acanthosis nigricans, a cutaneous marker of insulin resistance, was observed in 31.5% of PCOS cases, and androgenic alopecia was the least common feature, present in 19.4% of cases.

DISCUSSION

This study found an overall PCOS prevalence of 18.0% among young women aged 15–29 years using Rotterdam 2003 criteria, a figure that sits well within the wide range of 5% to 26% reported globally and is closely comparable to pooled estimates from South Asian populations(5,7). Our prevalence estimate is notably similar to the pooled meta-analytic figure of 17.74% reported among Indian adolescents using the same diagnostic framework, and is consistent with several community-based Indian studies reporting rates between 17% and 22.5% among young women and college students(8,9). This concordance lends external validity to our findings and reinforces the observation that South Asian populations, and Indian women in particular, may bear a disproportionately heavy burden of PCOS compared with East Asian or Caucasian populations, where Rotterdam-based prevalence is generally lower (approximately 5.6% to 11%)(7). Differences in genetic predisposition, particularly higher intrinsic insulin resistance among South Asians compared with their European counterparts, alongside the rapid nutritional transition occurring in urbanising regions, may partly explain this disparity(11). Consistent with prior literature, the prevalence of PCOS in our study varied markedly depending on the diagnostic criteria applied, ranging from 6.5% under the conservative NIH criteria to 18.0% under the more inclusive Rotterdam criteria. This pattern mirrors findings from earlier meta-analyses showing that Rotterdam-based estimates are typically two to three times higher than NIH-based estimates, since the Rotterdam framework captures phenotypes with ultrasonographic polycystic ovaries in the absence of clinical or biochemical hyperandrogenism(6,7). This methodological variability has important public health implications: studies and screening programmes that rely on differing criteria are not directly comparable, and policymakers should be cautious when interpreting prevalence figures without accounting for the diagnostic framework used.

The strong association observed between obesity and PCOS in our cohort (OR 5.74) is in keeping with the well-established bidirectional relationship between adiposity and androgen excess. Obesity, particularly central/visceral adiposity, promotes hyperinsulinaemia, which in turn stimulates ovarian theca cell androgen production and suppresses hepatic synthesis of sex hormone-binding globulin, increasing the bioavailability of free testosterone(11,13). Notably, insulin resistance has been documented in up to 80% of obese women with PCOS compared with only 30–40% of lean women with the condition, suggesting that while obesity is a major amplifying factor, PCOS can and does occur in lean phenotypes through alternative or primary ovarian/adrenal mechanisms(11). The significant association with sedentary lifestyle and high-calorie dietary patterns observed in our study further supports the role of modifiable lifestyle factors in disease expression, and is consistent with evidence that structured lifestyle modification, even modest weight reduction of 5–10%, can restore ovulatory function and improve the metabolic profile in affected women(12).

The significant association of family history with PCOS in our study (OR 3.16) corroborates the substantial heritable component of the syndrome that has been demonstrated in twin and familial aggregation studies, suggesting that first-degree relatives of affected women warrant closer surveillance and earlier screening(12). The clinical profile of our PCOS cases, dominated by menstrual irregularity (87.0%) and ultrasonographic polycystic morphology (75.0%), mirrors the typical adolescent and young-adult presentation described in the literature, where cutaneous markers such as acne and hirsutism are common but biochemical confirmation is essential given the overlap of isolated symptoms with normal pubertal physiology(10). The presence of acanthosis nigricans in nearly a third of our PCOS cases is a clinically useful bedside marker, as it correlates strongly with underlying insulin resistance and can prompt earlier metabolic screening even in resource-limited settings where biochemical assays are not readily available(13).

These findings carry important implications for clinical practice and public health policy. Given the high prevalence identified and the substantial proportion of asymptomatic or undiagnosed cases typically missed in hospital-based studies, school- and college-based screening programmes, alongside community health worker training to recognise early cutaneous and menstrual red flags, could facilitate earlier identification(9). Early diagnosis allows for timely initiation of lifestyle interventions before the secondary metabolic and cardiovascular sequelae of PCOS become established, with downstream benefits for fertility preservation and long-term chronic disease prevention(3,12). This study is, however, subject to certain limitations. As a cross-sectional design, it cannot establish temporal or causal relationships between identified risk factors and PCOS. The single-centre catchment area may limit generalisability to other populations with differing genetic and dietary profiles, and reliance on self-reported dietary and stress data introduces potential recall and reporting bias. Future longitudinal, multicentric studies incorporating detailed metabolic and hormonal profiling would help clarify causal pathways and evaluate the long-term impact of early intervention.

CONCLUSION

This study demonstrates that PCOS affects a substantial proportion of young women, with a prevalence of 18.0% using Rotterdam criteria, reflecting a considerable and likely under-recognised disease burden in this population. Obesity, sedentary lifestyle, unhealthy dietary patterns, high perceived stress, and a positive family history emerged as significant and largely modifiable or identifiable risk factors. Given that menstrual irregularity and cutaneous androgen excess were the most common clinical presentations, increased awareness among young women, families, and primary healthcare

providers is essential for timely recognition. Community- and institution-based screening, combined with structured lifestyle modification programmes targeting weight management and physical activity, should be prioritised as cost-effective strategies to reduce both the immediate reproductive and long-term metabolic consequences of PCOS in this vulnerable age group.

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