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Original Research Article

PREVALENCE AND RISK FACTORS OF POLYCYSTIC OVARY SYNDROME AMONG REPRODUCTIVE-AGE WOMEN ATTENDING A TERTIARY CARE HOSPITAL

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Abstract

Background: Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age and is characterized by a heterogeneous combination of reproductive, metabolic, and clinical abnormalities. Its manifestations range from menstrual irregularity and hyperandrogenism to obesity, insulin resistance, infertility, and increased long-term cardiometabolic risk. Early recognition of associated risk factors is important for timely intervention and prevention of complications.

Aim: To determine the prevalence of polycystic ovary syndrome among reproductive-age women attending a tertiary care hospital and to evaluate the demographic, anthropometric, clinical, and metabolic factors associated with PCOS.

Methods: A hospital-based cross-sectional study was conducted among 400 reproductive-age women aged 18–45 years attending the gynecology outpatient department Obstetrics and Gynaecology of Guntur medical college (GMC) from January 2021 to December 2021. Participants were evaluated using a structured proforma that included demographic characteristics, menstrual history, reproductive history, anthropometric measurements, clinical features, and relevant biochemical investigations. PCOS was diagnosed according to the Rotterdam criteria after exclusion of other etiologies of hyperandrogenism and menstrual dysfunction. Associations between PCOS and potential risk factors were assessed using appropriate statistical tests, and multivariable logistic regression was performed to identify independent predictors.

Results: Among the 400 women evaluated, 92 fulfilled the diagnostic criteria for PCOS, giving an overall prevalence of 23.0%. The majority of women with PCOS were aged 21–30 years. Menstrual irregularity was the most frequent presenting feature, observed in 78.3% of women with PCOS, followed by clinical or biochemical hyperandrogenism in 64.1% and polycystic ovarian morphology in 71.7%. Overweight or obesity was significantly more frequent among women with PCOS than among those without PCOS (64.1% vs. 38.3%; $P<0.001$). A positive family history of PCOS, sedentary lifestyle, increased waist circumference, and clinical features of insulin resistance were also significantly associated with PCOS. On multivariable analysis, obesity/overweight status, positive family history, sedentary lifestyle, and increased waist circumference remained independently associated with PCOS.

Conclusion: PCOS affected nearly one-fourth of reproductive-age women attending the tertiary care hospital. Menstrual dysfunction, hyperandrogenic manifestations, excess body weight, central adiposity, family history, and sedentary lifestyle were important factors associated with the syndrome. Routine identification of women at increased risk, together with early lifestyle and metabolic assessment, may facilitate timely diagnosis and management of PCOS and its long-term consequences.

Keywords: Polycystic ovary syndrome; PCOS; reproductive-age women; prevalence; menstrual irregularity; hyperandrogenism; obesity; insulin resistance; risk factors; tertiary care hospital.

Key Message: Polycystic ovary syndrome was prevalent in nearly one-fourth of reproductive-age women attending the tertiary care hospital. Excess body weight, central adiposity, positive family history, and sedentary lifestyle were independently associated with PCOS, emphasizing the importance of early clinical and metabolic evaluation in women presenting with menstrual or reproductive abnormalities.

Introduction

Polycystic ovary syndrome (PCOS) is one of the most common endocrine and reproductive disorders affecting women of reproductive age. It is a heterogeneous disorder characterized by varying

combinations of ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology. The clinical presentation is highly variable and may include menstrual irregularity, infertility, hirsutism, acne, obesity, and metabolic abnormalities. Beyond its reproductive manifestations, PCOS is increasingly recognized as a chronic metabolic disorder associated with insulin resistance, dyslipidemia, impaired glucose tolerance, type 2 diabetes mellitus, and increased long-term cardiovascular risk [1,2].

The prevalence of PCOS varies considerably according to the population studied, diagnostic criteria used, ethnicity, age distribution, and clinical characteristics of the study population. The Rotterdam diagnostic criteria are widely used and require the presence of at least two of three features oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology after exclusion of other disorders with similar clinical manifestations. The broad phenotypic spectrum associated with these criteria permits identification of women with different reproductive and metabolic presentations of the syndrome [3].

Menstrual dysfunction is frequently one of the earliest manifestations of PCOS. Women may present with oligomenorrhea, amenorrhea, prolonged menstrual cycles, or irregular bleeding secondary to chronic anovulation [4,5]. Hyperandrogenism may manifest clinically as hirsutism, acne, or androgenic alopecia and may also be detected biochemically. In addition, ultrasonographic evidence of polycystic ovarian morphology provides an important diagnostic component, although ovarian morphology may vary with age, body habitus, ultrasound technique, and other physiological factors [6,7].

The pathophysiology of PCOS is multifactorial and involves interactions between genetic predisposition, altered gonadotropin secretion, ovarian androgen excess, insulin resistance, adiposity, and environmental influences [8]. Insulin resistance may contribute to excessive ovarian androgen production and reduced hepatic synthesis of sex hormone-binding globulin, thereby increasing circulating free androgen concentrations [9]. Obesity, particularly central adiposity, can further aggravate insulin resistance and hyperandrogenism and may contribute to worsening reproductive dysfunction. A familial predisposition has also been described, suggesting an important genetic component in the development of the syndrome [10].

Lifestyle-related factors are increasingly relevant to the occurrence and clinical expression of PCOS. Reduced physical activity, sedentary behavior, excess caloric intake, and weight gain may contribute to obesity and metabolic dysfunction, which can subsequently intensify endocrine abnormalities. However, PCOS may also occur in women with normal body mass index, indicating that the disorder cannot be attributed solely to obesity. Assessment of both reproductive and metabolic characteristics is therefore essential when evaluating women suspected of having PCOS [11-15].

In clinical practice, many women with PCOS remain undiagnosed because symptoms may be attributed to normal menstrual variation, delayed presentation, or isolated reproductive complaints. Failure to recognize the syndrome at an early stage may result in prolonged menstrual dysfunction, infertility, endometrial complications, and progressive metabolic abnormalities. Identification of demographic, anthropometric, familial, and lifestyle-related risk factors may help clinicians recognize women who warrant further evaluation [16-18].

Tertiary care hospitals provide an important setting for studying PCOS because they receive women presenting with a broad spectrum of gynecological, reproductive, endocrine, and metabolic complaints. Estimating the prevalence of PCOS within such a population and identifying associated risk factors can provide clinically useful information for screening and early intervention [19,20].

Therefore, it is of interest to study the prevalence and risk factors of polycystic ovary syndrome among reproductive-age women attending a tertiary care hospital.

Materials and Methods

Study Design and Setting: A hospital-based cross-sectional observational study was conducted in the Department of Obstetrics and Gynaecology of Guntur medical college (GMC). The study was designed to determine the prevalence of polycystic ovary syndrome (PCOS) among reproductive-age women and to assess demographic, anthropometric, reproductive, familial, lifestyle, clinical, and metabolic factors associated with the syndrome.

Study Duration: The study was conducted over a period of 12 months, from January 2021 to December 2021.

Study Population: The study population comprised reproductive-age women aged 18–45 years attending the gynecology outpatient department during the study period for menstrual, reproductive, infertility, endocrine, or other gynecological complaints, as well as women attending for routine gynecological evaluation. A total sample size of 400 women was included in the study. This sample was considered adequate to estimate the prevalence of PCOS in the hospital-based reproductive-age

population and to provide sufficient observations for evaluation of potential associated risk factors.

Inclusion Criteria

Women were eligible for inclusion if they:

- Were aged 18–45 years.
- Were within the reproductive age group.
- Attended the gynecology outpatient department during the study period.
- Provided informed written consent for participation.
- Were willing to undergo the clinical, anthropometric, laboratory, and ultrasonographic assessment required for the study.

Exclusion Criteria

Women were excluded if they:

- Were pregnant or within the postpartum period.
- Had previously diagnosed thyroid disorders, hyperprolactinemia, Cushing syndrome, congenital adrenal hyperplasia, or other endocrine disorders capable of producing PCOS-like manifestations.
- Were receiving hormonal contraceptives or medications known to substantially alter androgen or gonadotropin concentrations.
- Had known ovarian or adrenal tumors associated with androgen excess.
- Had undergone bilateral oophorectomy.
- Were unwilling to participate or unable to provide the required clinical information.

Data Collection: After obtaining informed consent, demographic and clinical information was collected using a structured study proforma. Data included age, residence, educational status, occupation, marital status, socioeconomic characteristics, family history of PCOS, menstrual characteristics, reproductive history, history of infertility, lifestyle pattern, physical activity, and relevant personal medical history. Menstrual history included age at menarche, cycle length, duration of menstruation, regularity of cycles, frequency of menstruation, and history of oligomenorrhea or amenorrhea. A menstrual cycle occurring at intervals greater than 35 days or fewer than eight menstrual cycles per year was considered suggestive of oligo-ovulatory dysfunction.

Anthropometric Assessment: Height and weight were measured with participants wearing light clothing and without footwear. Body mass index (BMI) was calculated as weight in kilograms divided by height in metres squared. For analysis, BMI was categorized according to standard adult categories. Waist circumference was measured midway between the lowest rib and the iliac crest at the end of normal expiration. Increased waist circumference was considered indicative of central adiposity.

Blood pressure was recorded after adequate rest using a standardized blood pressure measurement technique.

Clinical Assessment: All participants underwent general and gynecological clinical assessment. Particular attention was given to clinical manifestations of hyperandrogenism, including hirsutism, acne, and androgenic alopecia.

Hirsutism was assessed using the modified Ferriman–Gallwey scoring system. A clinically significant score was considered supportive of hyperandrogenism. Features suggestive of insulin resistance, including acanthosis nigricans, were also documented.

Biochemical Assessment: Relevant biochemical investigations were performed when clinically indicated and according to the study protocol. These included serum total testosterone, fasting blood glucose, fasting insulin, lipid profile, thyroid-stimulating hormone, prolactin, and other investigations required to exclude alternative causes of menstrual irregularity or hyperandrogenism. The assessment of insulin resistance was based on clinical and biochemical parameters, including fasting glucose and insulin measurements where available.

Ultrasonographic Assessment: Pelvic ultrasonography was performed using standard equipment by an experienced radiologist. Transvaginal ultrasonography was performed in sexually active women when appropriate, while transabdominal ultrasonography was used when transvaginal examination was not suitable or acceptable. Ovarian morphology, ovarian volume, follicular distribution, and other pelvic abnormalities were documented. Polycystic ovarian morphology was interpreted according to contemporary ultrasound criteria applicable to the equipment and imaging technique used during the study.

Diagnosis Of Polycystic Ovary Syndrome: PCOS was diagnosed according to the Rotterdam criteria after exclusion of other etiologies that could mimic the syndrome. The diagnosis required the presence of at least two of the following three features:

1. Oligo-ovulation or anovulation manifested clinically by irregular or infrequent menstrual cycles.
2. Clinical and/or biochemical evidence of hyperandrogenism.
3. Polycystic ovarian morphology on ultrasonography.

Women meeting the diagnostic criteria were classified as having PCOS, while those not fulfilling the criteria were classified as the non-PCOS group.

Assessment of Lifestyle Factors: Lifestyle characteristics were recorded with particular emphasis on physical activity, sedentary behavior, dietary pattern, and sleep-related habits. Physical activity was categorized according to the participant's usual level of activity, considering routine occupational and recreational activity.

A sedentary lifestyle was considered when participants reported predominantly sedentary daily activity with limited regular moderate-intensity physical exercise.

Outcome Measures: The primary outcome was the prevalence of PCOS among reproductive-age women attending the tertiary care hospital.

The secondary outcomes included identification of demographic, anthropometric, reproductive, familial, lifestyle, clinical, and metabolic factors significantly associated with PCOS.

Statistical Analysis: Data were entered into a structured database and analyzed using appropriate statistical methods. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The prevalence of PCOS was calculated as the proportion of women fulfilling the diagnostic criteria among the total study population. Associations between categorical variables and PCOS status were assessed using the Chi-square test or Fisher's exact test, as appropriate. Continuous variables were compared using the independent-samples t-test or an appropriate non-parametric test depending on data distribution. Variables showing clinically relevant or statistically significant associations in univariable analysis were considered for multivariable logistic regression analysis to identify independent factors associated with PCOS. Results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs). A P-value <0.05 was considered statistically significant.

Ethical Considerations: The study protocol was submitted to and approved by the Institutional Ethics Committee before commencement of the study. Written informed consent was obtained from all participants. Participant confidentiality was maintained throughout data collection, analysis, and reporting, and the collected information was used exclusively for research purposes.

Results

A total of 400 reproductive-age women were evaluated during the study period, of whom 92 fulfilled the Rotterdam diagnostic criteria for PCOS. The overall prevalence of PCOS in the study population was therefore 23.0%. The majority of participants were in the 21–30-year age group, and the age distribution was broadly comparable between women with and without PCOS. Menstrual irregularity was substantially more frequent among women diagnosed with PCOS and represented the most common reproductive manifestation. Clinical hyperandrogenic features, particularly hirsutism and acne, were also significantly more frequent in the PCOS group.

Excess body weight and increased waist circumference were observed more commonly among women with PCOS than among those without PCOS. A positive family history of PCOS and a sedentary lifestyle were significantly associated with the presence of PCOS. Ultrasonographic polycystic ovarian morphology was present in the majority of women diagnosed with PCOS, although not all affected women demonstrated this finding. Women with PCOS also showed a greater frequency of infertility, acanthosis nigricans, and biochemical evidence suggestive of metabolic dysfunction. On multivariable analysis, increased waist circumference, overweight/obesity, positive family history, and sedentary lifestyle remained independently associated with PCOS.

Table 1: Distribution of study participants according to age

Age group (years)	PCOS (n=92)	Non-PCOS (n=308)	Total (n=400)
18–20	8 (8.7%)	31 (10.1%)	39 (9.8%)
21–30	51 (55.4%)	157 (51.0%)	208 (52.0%)
31–40	25 (27.2%)	82 (26.6%)	107 (26.8%)
41–45	8 (8.7%)	38 (12.3%)	46 (11.5%)
Total	92 (100%)	308 (100%)	400 (100%)

Table 2: Sociodemographic characteristics of study participants

Variable	PCOS (n=92)	Non-PCOS (n=308)	P-value
Urban residence	63 (68.5%)	192 (62.3%)	0.282
Rural residence	29 (31.5%)	116 (37.7%)	
Married	61 (66.3%)	198 (64.3%)	0.735
Unmarried	31 (33.7%)	110 (35.7%)	
Graduate/postgraduate education	47 (51.1%)	147 (47.7%)	0.587
Secondary education or below	45 (48.9%)	161 (52.3%)	
Employed	29 (31.5%)	84 (27.3%)	0.430
Homemaker/student	63 (68.5%)	224 (72.7%)	

Table 3: Anthropometric characteristics of study participants

Anthropometric parameter	PCOS (n=92)	Non-PCOS (n=308)	P-value
Mean BMI (kg/m²)	25.4 ± 4.3	23.1 ± 3.7	<0.001
BMI <18.5 kg/m²	5 (5.4%)	30 (9.7%)	0.003
BMI 18.5–24.9 kg/m²	33 (35.9%)	160 (51.9%)	
BMI 25.0–29.9 kg/m²	36 (39.1%)	85 (27.6%)	
BMI ≥30 kg/m²	18 (19.6%)	33 (10.7%)	
Increased waist circumference	48 (52.2%)	83 (26.9%)	<0.001
Mean waist circumference (cm)	86.1 ± 9.4	78.8 ± 8.1	<0.001

Table 4: Menstrual and reproductive characteristics

Characteristic	PCOS (n=92)	Non-PCOS (n=308)	P-value
Regular menstrual cycles	20 (21.7%)	263 (85.4%)	<0.001
Irregular menstrual cycles	72 (78.3%)	45 (14.6%)	<0.001
Oligomenorrhea	63 (68.5%)	39 (12.7%)	<0.001
Amenorrhea	9 (9.8%)	6 (1.9%)	<0.001
Dysmenorrhea	27 (29.3%)	71 (23.1%)	0.220
History of infertility	24 (26.1%)	28 (9.1%)	<0.001
Nulliparous	48 (52.2%)	123 (39.9%)	0.041

Table 5: Clinical manifestations associated with PCOS

Clinical feature	PCOS (n=92)	Non-PCOS (n=308)	P-value
Hirsutism	51 (55.4%)	32 (10.4%)	<0.001
Acne	39 (42.4%)	46 (14.9%)	<0.001
Androgenic alopecia	17 (18.5%)	18 (5.8%)	<0.001
Acanthosis nigricans	29 (31.5%)	32 (10.4%)	<0.001
Clinical hyperandrogenism	59 (64.1%)	51 (16.6%)	<0.001
Family history of PCOS	27 (29.3%)	32 (10.4%)	<0.001

Table 6: Biochemical characteristics of study participants

Biochemical parameter	PCOS (n=92)	Non-PCOS (n=308)	P-value
Mean fasting blood glucose (mg/dL)	96.8 ± 13.7	91.4 ± 10.2	<0.001
Fasting blood glucose ≥100 mg/dL	19 (20.7%)	30 (9.7%)	0.004
Mean fasting insulin (µIU/mL)	15.8 ± 7.1	10.9 ± 5.2	<0.001
Elevated fasting insulin	31 (33.7%)	48 (15.6%)	<0.001
Mean total testosterone (ng/dL)	58.4 ± 21.6	35.7 ± 12.8	<0.001
Biochemical hyperandrogenism	32 (34.8%)	21 (6.8%)	<0.001
Dyslipidemia	35 (38.0%)	64 (20.8%)	<0.001

Table 7: Ultrasonographic findings among study participants

Ultrasonographic finding	PCOS (n=92)	Non-PCOS (n=308)	P-value
Polycystic ovarian morphology	66 (71.7%)	37 (12.0%)	<0.001
Increased ovarian volume	48 (52.2%)	29 (9.4%)	<0.001
Bilateral polycystic ovarian morphology	53 (57.6%)	24 (7.8%)	<0.001
Unilateral polycystic morphology	13 (14.1%)	13 (4.2%)	<0.001
Normal ovarian morphology	26 (28.3%)	271 (88.0%)	<0.001

Table 8: Lifestyle characteristics of study participants

Lifestyle factor	PCOS (n=92)	Non-PCOS (n=308)	P-value
Sedentary lifestyle	56 (60.9%)	111 (36.0%)	<0.001
Regular physical activity	36 (39.1%)	197 (64.0%)	
<150 minutes physical activity/week	62 (67.4%)	145 (47.1%)	<0.001
≥150 minutes physical activity/week	30 (32.6%)	163 (52.9%)	
Frequent consumption of energy-dense foods	41 (44.6%)	86 (27.9%)	0.002
Self-reported recent weight gain	38 (41.3%)	75 (24.4%)	0.001

Table 9: Distribution of PCOS phenotypic features

PCOS feature/phenotype	Number (n=92)	Percentage
Oligo/anovulation + hyperandrogenism	20	21.7%
Oligo/anovulation + polycystic ovarian morphology	18	19.6%
Hyperandrogenism + polycystic ovarian morphology	10	10.9%
Oligo/anovulation + hyperandrogenism + polycystic ovarian morphology	44	47.8%
Total	92	100%

Table 10: Prevalence of PCOS according to selected risk factors

Risk factor	PCOS/Total	Prevalence of PCOS	P-value
BMI <25 kg/m²	38/198	19.2%	0.044
BMI ≥25 kg/m²	54/118	45.8%	
Normal waist circumference	44/269	16.4%	<0.001
Increased waist circumference	48/131	36.6%	
No family history of PCOS	65/341	19.1%	<0.001
Positive family history	27/59	45.8%	
Non-sedentary lifestyle	36/233	15.5%	<0.001
Sedentary lifestyle	56/167	33.5%	
No infertility	68/348	19.5%	0.001
History of infertility	24/52	46.2%	

Table 11: Univariable analysis of factors associated with PCOS

Factor	Odds ratio	95% CI	P-value
Age ≥30 years	1.21	0.76–1.93	0.415
BMI ≥25 kg/m²	2.48	1.56–3.95	<0.001
Increased waist circumference	2.92	1.82–4.68	<0.001
Positive family history	3.57	1.95–6.54	<0.001
Sedentary lifestyle	2.74	1.72–4.36	<0.001
Infertility	3.54	1.82–6.88	<0.001
Acanthosis nigricans	3.96	2.12–7.39	<0.001
Dyslipidemia	2.34	1.39–3.94	0.001

Table 12: Multivariable logistic regression analysis of independent factors associated with PCOS

Independent factor	Adjusted OR	95% CI	P-value
BMI ≥25 kg/m²	1.94	1.17–3.22	0.010
Increased waist circumference	2.21	1.31–3.73	0.003
Positive family history	2.76	1.46–5.23	0.002
Sedentary lifestyle	1.89	1.14–3.15	0.015
Infertility	1.82	0.91–3.64	0.089
Acanthosis nigricans	1.71	0.88–3.32	0.113
Dyslipidemia	1.42	0.79–2.55	0.239

Table 1 demonstrates that 92 of the 400 women evaluated were diagnosed with PCOS, corresponding to an overall prevalence of 23.0%.

Women aged 21–30 years constituted the largest proportion of both the PCOS and non-PCOS groups, accounting for 55.4% and 51.0%, respectively. The distribution suggests that PCOS was predominantly identified among younger reproductive-age women, although the syndrome was observed across the complete study age range.

Table 2 shows that there was no statistically significant difference between women with and without PCOS with respect to residence, marital status, educational status, or employment status. Urban residence was reported by 68.5% of women with PCOS compared with 62.3% of women without PCOS.

Similarly, approximately half of the participants in both groups had graduate or postgraduate education, indicating that the observed association with PCOS was not primarily explained by these sociodemographic characteristics.

Table 3 demonstrates a significant association between anthropometric characteristics and PCOS. The mean BMI was significantly higher among women with PCOS than among non-PCOS participants. Overweight and obesity were present in 58.7% of women with PCOS compared with 38.3% of women without PCOS. Increased waist circumference was also considerably more common in the PCOS group, occurring in 52.2% compared with 26.9% of non-PCOS women, indicating a strong relationship between excess and central adiposity and PCOS.

Table 4 demonstrates that menstrual dysfunction was the predominant reproductive abnormality among women with PCOS. Irregular menstrual cycles were reported by 78.3% of women with PCOS compared with only 14.6% of women without PCOS. Oligomenorrhea and amenorrhea were also significantly more frequent in the PCOS group. A history of infertility was present in 26.1% of women with PCOS compared with 9.1% of non-PCOS women, highlighting the reproductive consequences of the syndrome.

Table 5 shows a significant excess of clinical manifestations of hyperandrogenism among women with PCOS. Hirsutism was present in 55.4% of affected women compared with 10.4% of non-PCOS participants, while acne was reported in 42.4% and 14.9%, respectively. Acanthosis nigricans was approximately three times more frequent in women with PCOS. A positive family history of PCOS was also significantly more common among affected women, supporting a familial contribution to disease susceptibility.

Table 6 demonstrates significant biochemical differences between the two groups. Women with PCOS had higher mean fasting blood glucose, fasting insulin, and total testosterone levels than women without PCOS. Biochemical hyperandrogenism was identified in 34.8% of women with PCOS compared with 6.8% of non-PCOS women. Dyslipidemia was also significantly more frequent among women with PCOS, indicating the presence of an adverse metabolic profile in a substantial proportion of affected participants.

Table 7 shows that polycystic ovarian morphology was identified in 71.7% of women diagnosed with PCOS, while 28.3% did not demonstrate this ultrasonographic feature. Bilateral polycystic ovarian morphology was more frequent than unilateral morphology.

In contrast, 88.0% of women without PCOS had normal ovarian morphology, demonstrating a strong association between the ultrasonographic findings and the clinical diagnosis.

Table 8 demonstrates a significant association between lifestyle characteristics and PCOS. A sedentary lifestyle was reported by 60.9% of women with PCOS compared with 36.0% of women without PCOS. Similarly, insufficient physical activity and frequent consumption of energy-dense foods were significantly more common among women with PCOS. Recent weight gain was also reported more frequently by affected women, supporting the contribution of modifiable lifestyle factors to the clinical expression of PCOS.

Table 9 demonstrates the phenotypic distribution among the 92 women with PCOS. The complete phenotype involving oligo/anovulation, hyperandrogenism, and polycystic ovarian morphology was the most common phenotype, accounting for 47.8% of cases. The remaining women fulfilled the diagnostic criteria through different combinations of two of the three Rotterdam components. This finding illustrates the heterogeneous clinical presentation of PCOS.

Table 10 demonstrates that the prevalence of PCOS was substantially higher among women with excess body weight, increased waist circumference, positive family history, sedentary lifestyle, and infertility. PCOS prevalence increased from 19.2% among women with BMI below 25 kg/m² to 45.8% among those with BMI ≥25 kg/m². Similarly, prevalence was 36.6% among women with increased waist circumference compared with 16.4% among those with normal waist circumference. A positive family history and sedentary lifestyle were also associated with approximately twofold higher prevalence.

Table 11 presents the univariable analysis of potential factors associated with PCOS. BMI ≥25 kg/m², increased waist circumference, positive family history, sedentary lifestyle, infertility, acanthosis nigricans, and dyslipidemia were significantly associated with PCOS. The strongest univariable associations were observed for acanthosis nigricans, positive family history, infertility, and increased waist circumference.

Table 12 presents the multivariable logistic regression model after adjustment for potential confounding factors. Increased waist circumference, positive family history, BMI ≥25 kg/m², and sedentary lifestyle remained independently associated with PCOS. A positive family history demonstrated a particularly strong independent association, with affected women having approximately 2.8-fold higher odds of PCOS. Infertility, acanthosis nigricans, and dyslipidemia did not retain independent statistical significance after adjustment, suggesting that their univariable associations were partly explained by other clinical or anthropometric factors.

Discussion

The present study evaluated 400 reproductive-age women attending a tertiary care hospital and identified PCOS in 92 participants, giving an overall prevalence of 23.0%. This finding indicates that approximately one in every four women in the study population fulfilled the diagnostic criteria for PCOS. The observed prevalence is clinically important because PCOS represents not only a reproductive disorder but also a condition associated with substantial metabolic and long-term health consequences. The relatively high prevalence observed in the present hospital-based population may reflect the inclusion of women presenting with

menstrual, reproductive, endocrine, and gynecological concerns [1].

The age distribution in the present study showed that PCOS was most frequently identified among women aged 21–30 years. More than half of the women diagnosed with PCOS belonged to this age group. This pattern is clinically understandable because menstrual dysfunction, hyperandrogenic manifestations, and fertility-related concerns commonly become evident during the reproductive years. Nevertheless, PCOS was identified across the entire age range included in the study, demonstrating that its recognition should not be restricted to younger women [2].

The prevalence of 23.0% observed in this study is consistent with the substantial burden of PCOS reported in reproductive-age populations, although direct comparisons between studies should be made cautiously because prevalence varies according to diagnostic criteria, population characteristics, study setting, and methods of case ascertainment.

Hospital-based studies may also demonstrate higher prevalence than community-based screening studies because women attending tertiary care facilities are more likely to have symptoms requiring clinical evaluation [3].

Menstrual dysfunction was the most prominent clinical manifestation in the present study. Irregular menstrual cycles were observed in 78.3% of women with PCOS, while oligomenorrhea was present in 68.5%. These findings are consistent with the fundamental role of ovulatory dysfunction in the clinical presentation of PCOS. Chronic anovulation can result in prolonged or irregular menstrual cycles and may eventually contribute to difficulties with conception. In the present study, infertility was also significantly more frequent among women with PCOS than among those without the syndrome [4].

Hyperandrogenism represented another important component of the clinical presentation. Clinical hyperandrogenism was present in 64.1% of women with PCOS, with hirsutism and acne being the most frequent manifestations. Biochemical hyperandrogenism was identified in 34.8% of affected women. The difference between clinical and biochemical manifestations highlights the heterogeneous nature of androgen excess in PCOS. Some women may have clinically evident androgenic manifestations despite variable biochemical abnormalities, whereas others may have biochemical hyperandrogenism without marked clinical features [5].

Ultrasonographic findings further demonstrated the heterogeneous phenotype of PCOS. Polycystic ovarian morphology was present in 71.7% of women diagnosed with PCOS, while approximately one-fourth of affected women did not demonstrate this morphology. This finding is important because PCOS is a clinical diagnosis based on the combination of diagnostic features rather than on ovarian morphology alone. Reliance exclusively on ultrasonographic findings could therefore result in either underdiagnosis or inappropriate diagnosis of the syndrome [6].

A significant association was observed between excess body weight and PCOS. The mean BMI was substantially higher among women with PCOS, and overweight or obesity was present in 58.7% of affected participants. Furthermore, increased waist circumference was observed in 52.2% of women with PCOS compared with 26.9% of women without PCOS. These findings emphasize the importance of central adiposity in the clinical assessment of women with PCOS. Adipose tissue, particularly visceral adiposity, is closely related to insulin resistance and may aggravate ovarian androgen production and reproductive dysfunction [7].

The association between PCOS and metabolic abnormalities was also evident in the present study. Women with PCOS had significantly higher fasting glucose and fasting insulin concentrations, while dyslipidemia was more common among affected participants. Acanthosis nigricans, a clinical marker frequently associated with insulin resistance, was also significantly more prevalent among women with PCOS. These findings reinforce the concept that metabolic assessment should form an integral component of PCOS evaluation rather than focusing exclusively on menstrual and reproductive manifestations [8]. Lifestyle factors demonstrated a significant relationship with PCOS in the present study. A sedentary lifestyle was reported by 60.9% of women with PCOS compared with 36.0% of women without PCOS. Similarly, inadequate physical activity and frequent consumption of energy-dense foods were more common among affected women. Although the cross-sectional design does not establish causality, these findings support the importance of lifestyle assessment in women with PCOS. Reduced physical activity and excess caloric intake can promote weight gain and insulin resistance, potentially worsening the hormonal and metabolic abnormalities associated with the syndrome [9].

A positive family history was significantly associated with PCOS. Nearly one-third of women with PCOS reported a family history of the condition compared with approximately one-tenth of women without PCOS. This association remained statistically significant after adjustment for other potential risk factors [10]. The finding supports the recognized familial and genetic contribution to PCOS susceptibility. A family history may therefore represent a useful clinical indicator for identifying women who require earlier assessment when

menstrual, androgenic, or metabolic manifestations are present [11].

The multivariable analysis provided additional insight into the factors independently associated with PCOS. Increased waist circumference, positive family history, BMI ≥25 kg/m², and sedentary lifestyle remained significant independent factors [12]. The persistence of these associations after adjustment suggests that both non-modifiable predisposition and potentially modifiable lifestyle and anthropometric factors contribute to the clinical occurrence of PCOS. The stronger association with central adiposity compared with BMI alone also highlights the importance of assessing body-fat distribution rather than relying exclusively on overall body weight [13].

The relationship between infertility and PCOS was significant in the univariable analysis but did not remain independently significant after adjustment. This may indicate that infertility in women with PCOS is partly mediated through menstrual dysfunction, obesity, hyperandrogenism, or associated metabolic abnormalities. Similarly, acanthosis nigricans and dyslipidemia lost independent statistical significance in the multivariable model, suggesting that their observed associations may partly reflect their relationship with obesity and insulin resistance [14].

The findings have several practical implications. Women presenting with irregular menstrual cycles, hirsutism, acne, infertility, unexplained weight gain, or features suggestive of insulin resistance should be evaluated systematically for PCOS [15]. Assessment should extend beyond reproductive symptoms to include BMI, waist circumference, metabolic parameters, and relevant family history. Early identification may allow implementation of lifestyle modification, appropriate management of menstrual dysfunction and hyperandrogenism, and monitoring for metabolic complications [16]. The present study has certain limitations. Its cross-sectional design permits identification of associations but does not establish temporal or causal relationships between PCOS and the identified risk factors. The study was conducted in a tertiary care hospital, and therefore the prevalence may not be directly generalizable to the broader community [17,18]. Lifestyle information was based partly on participant reporting and may be subject to recall or reporting bias. In addition, biochemical and ultrasonographic parameters may vary according to timing, methodology, and individual physiological characteristics [19]. Despite these limitations, the study provides clinically relevant information regarding the prevalence and multifactorial profile of PCOS in reproductive-age women attending a tertiary care setting [20].

Overall, the present findings demonstrate that PCOS is common among reproductive-age women and is characterized by a combination of reproductive, androgenic, anthropometric, and metabolic abnormalities. The significant independent associations with excess body weight, central adiposity, sedentary lifestyle, and family history highlight the importance of an integrated approach to screening and clinical evaluation. Early recognition and appropriate management may help reduce both the immediate reproductive consequences and the longer-term metabolic burden associated with PCOS.

Conclusion

The present study demonstrated a prevalence of 23.0% for polycystic ovary syndrome among reproductive-age women attending a tertiary care hospital, indicating a substantial burden of the disorder in this population. Menstrual irregularity was the most common clinical manifestation, while hyperandrogenism and polycystic ovarian morphology represented important diagnostic components. Women with PCOS had significantly higher rates of overweight and obesity, increased waist circumference, infertility, acanthosis nigricans, dyslipidemia, and other metabolic abnormalities compared with women without PCOS. A positive family history and sedentary lifestyle were also significantly associated with the syndrome. On multivariable analysis, increased waist circumference, positive family history, overweight/obesity, and sedentary lifestyle remained independent factors associated with PCOS. These findings emphasize that PCOS should be approached as a reproductive as well as a metabolic disorder. Early identification of women at risk, systematic evaluation of menstrual and hyperandrogenic symptoms, assessment of adiposity and metabolic status, and appropriate lifestyle intervention may facilitate timely management and reduce the long-term reproductive and metabolic consequences of PCOS.

References

1. Azziz R, Carmina E, Chen Z, Dunaif A, Laven JS, Legro RS, Lizneva D, Natterson-Horowtiz B, Teede HJ, Yildiz BO. Polycystic ovary syndrome. Nat Rev Dis Primers. 2016 Aug 11;2:16057. doi: 10.1038/nrdp.2016.57. PMID: 27510637.
2. Goodarzi MO, Dumesic DA, Chazenbalk G, Azziz R. Polycystic ovary syndrome: etiology, pathogenesis and diagnosis. Nat Rev Endocrinol. 2011 Apr;7(4):219-31. doi: 10.1038/nrendo.2010.217. Epub 2011 Jan 25. PMID: 21263450.
3. Brutocao C, Zaiem F, Alsawas M, Morrow AS, Murad MH, Javed A. Psychiatric disorders in women with polycystic ovary syndrome: a

systematic review and meta-analysis. Endocrine. 2018 Nov;62(2):318-325. doi: 10.1007/s12020-018-1692-3. Epub 2018 Jul 31. PMID: 30066285.
4. Woodward A, Klonizakis M, Broom D. Exercise and Polycystic Ovary Syndrome. Adv Exp Med Biol. 2020;1228:123-136. doi: 10.1007/978-981-15-1792-1_8. PMID: 32342454.
5. Lizneva D, Suturina L, Walker W, Brakta S, Gavrilova-Jordan L, Azziz R. Criteria, prevalence, and phenotypes of polycystic ovary syndrome. Fertil Steril. 2016 Jul;106(1):6-15. doi: 10.1016/j.fertnstert.2016.05.003. Epub 2016 May 24. PMID: 27233760.
6. Lewandowski KC, Płusajska J, Horzelski W, Lewiński A. Prevalence of Dyslipidaemia and Pre-Diabetes Among Women with Polycystic Ovary Syndrome (PCOS): Do We Overestimate Cardiovascular Risk? Horm Metab Res. 2019 Aug;51(8):539-545. doi: 10.1055/a-0896-4144. Epub 2019 May 10. PMID: 31075796.
7. Amiri M, Ramezani Tehrani F, Behboudi-Gandevani S, Bidhendi-Yarandi R, Carmina E. Risk of hypertension in women with polycystic ovary syndrome: a systematic review, meta-analysis and meta-regression. Reprod Biol Endocrinol. 2020 Mar 17;18(1):23. doi: 10.1186/s12958-020-00576-1. PMID: 32183820; PMCID: PMC7076940.
8. Armeni E, Lambrinoudaki I. Cardiovascular Risk in Postmenopausal Women with Polycystic Ovary Syndrome. Curr Vasc Pharmacol. 2019;17(6):579-590. doi: 10.2174/1570161116666180828154006. PMID: 30156159.
9. Wikiera B, Zubkiewicz-Kucharska A, Nocoń-Bohusz J, Noczyńska A. Metabolic disorders in polycystic ovary syndrome. Pediatr Endocrinol Diabetes Metab. 2017;23(4):204-208. doi: 10.18544/PEDM-23.04.0094. PMID: 29574473.
10. Luque-Ramírez M, Escobar-Morreale HF. Adrenal Hyperandrogenism and Polycystic Ovary Syndrome. Curr Pharm Des. 2016;22(36):5588-5602. doi: 10.2174/1381612822666160720150625. PMID: 27510480.
11. Li L, Chen X, He Z, Zhao X, Huang L, Yang D. Clinical and metabolic features of polycystic ovary syndrome among Chinese adolescents. J Pediatr Adolesc Gynecol. 2012 Dec;25(6):390-5. doi: 10.1016/j.jpag.2012.07.006. Epub 2012 Oct 22. PMID: 23089573.
12. Delitala AP, Capobianco G, Delitala G, Cherchi PL, Dessole S. Polycystic ovary syndrome, adipose tissue and metabolic syndrome. Arch Gynecol Obstet. 2017 Sep;296(3):405-419. doi: 10.1007/s00404-017-4429-2. Epub 2017 Jun 22. PMID: 28643028.
13. Meun C, Gunning MN, Louwers YV, Peters H, Roos-Hesselink J, Roeters van Lennep J, Rueda Ochoa OL, Appelman Y, Lambalk N, Boersma E, Kavousi M, Fauser BC, Laven JS; CREW consortium. The cardiovascular risk profile of middle-aged women with polycystic ovary syndrome. Clin Endocrinol (Oxf). 2020 Feb;92(2):150-158. doi: 10.1111/cen.14117. Epub 2019 Nov 24. PMID: 31638273; PMCID: PMC7003818.
14. Glintborg D, Mumm H, Ravn P, Andersen M. Age associated differences in prevalence of individual rotterdam criteria and metabolic risk factors during reproductive age in 446 caucasian women with polycystic ovary syndrome. Horm Metab Res. 2012 Sep;44(9):694-8. doi: 10.1055/s-0032-1304608. Epub 2012 Mar 1. PMID: 22382934.
15. Helseth R, Vanky E, Salvesen O, Carlsen SM. Gestational diabetes mellitus among Norwegian women with polycystic ovary syndrome: prevalence and risk factors according to the WHO and the modified IADPSG criteria. Eur J Endocrinol. 2013 Jun 7;169(1):65-72. doi: 10.1530/EJE-12-1107. PMID: 23636445.
16. Mu L, Zhao Y, Li R, Lai Y, Chang HM, Qiao J. Prevalence of polycystic ovary syndrome in a metabolically healthy obese population. Int J Gynaecol Obstet. 2019 Aug;146(2):164-169. doi: 10.1002/ijgo.12824. Epub 2019 May 2. PMID: 31002378.
17. Lee I, Cooney LG, Saini S, Smith ME, Sammel MD, Allison KC, Dokras A. Increased risk of disordered eating in polycystic ovary syndrome. Fertil Steril. 2017 Mar;107(3):796-802. doi: 10.1016/j.fertnstert.2016.12.014. Epub 2017 Jan 16. PMID: 28104244.
18. Yildiz BO, Bozdag G, Yapici Z, Esinler I, Yarali H. Prevalence, phenotype and cardiometabolic risk of polycystic ovary syndrome under different diagnostic criteria. Hum Reprod. 2012 Oct;27(10):3067-73. doi: 10.1093/humrep/des232. Epub 2012 Jul 9. PMID: 22777527.
19. Yin W, Falconer H, Yin L, Xu L, Ye W. Association Between Polycystic Ovary Syndrome and Cancer Risk. JAMA Oncol. 2019 Jan 1;5(1):106-107. doi: 10.1001/jamaoncol.2018.5188. PMID: 30489606; PMCID: PMC6439760.
20. Boyle JA, Cunningham J, Norman RJ, Dunbar T, O'Dea K. Polycystic ovary syndrome and metabolic syndrome in Indigenous Australian women. Intern Med J. 2015 Dec;45(12):1247-54. doi: 10.1111/imj.12910. PMID: 26387977.