



PREVALENCE AND IMPACT OF POLYCYSTIC OVARY SYNDROME (PCOS) ON FERTILITY AND METABOLIC HEALTH IN WOMEN.

Zainab Pirzada¹, Mah Rukh², Fozia Amin^{3*}, Asma Qadir⁴

¹Consultant Gynecologist Khalifa Gulnawaz Teaching Hospital MTI Bannu

²Consultant Gynecologist Frontier Corps Teaching Hospital Peshawar.

^{3*}Consultant Gynecologist Khalifa Gulnawaz Teaching Hospital MTI Bannu

⁴Consultant Gynecologist Type D Hospital Latamber Karak

***Corresponding Author:** Fozia Amin

Email: Kfozia151@gmail.com

ABSTRACT

Background: the prevalent endocrine disorders in women of reproductive age, and in Women especially, is Polycystic Ovary Syndrome (PCOS). It is hyperandrogenism, oligo-anovulation, and polycystic ovary. PCOS is a leading cause of infertility and also it exposes individuals to the risk of metabolic complications such as insulin insensitivity, obesity, and dyslipidemia, which affect long-term health outcomes.

Objectives: to assess the incidence of PCOS among Women and the effects of the disease on fertility and metabolic health, including reproductive dysfunction and related metabolic disorders.

Study design: A cross-sectional study.

Place and duration of study: Department Of Gynae-Obs Khalifa Gulnawaz Hospital Bannu From Jan 2024 To March 2024

Methods: This cross-sectional study included 100 women aged 18–30 years who visited a tertiary care hospital. Participants were assessed based on the Rotterdam criteria for the diagnosis of polycystic ovary syndrome (PCOS). Data on menstrual abnormalities, fertility, and metabolic variables were collected. Laboratory tests included fasting glucose, insulin levels, and lipid profiles. Statistical analysis was performed using SPSS, and mean values, standard deviations, and p-values for significant associations were calculated.

Results: A total of 100 women with polycystic ovary syndrome (PCOS) were studied, with an average age of 24.6 ± 3.2 years. Women with PCOS had significantly higher body mass index (BMI) ($p = 0.002$) and waist-to-hip ratio ($p = 0.01$) compared to controls. Insulin resistance was observed in 41% of PCOS cases ($p = 0.001$). Menstrual abnormalities were reported in 78% of affected women, with 35% experiencing infertility ($p = 0.004$). Dyslipidemia was significantly more prevalent in the PCOS group ($p = 0.03$). These findings underscore that PCOS is a dual burden, affecting both reproductive and metabolic health in women.

Conclusion: The analysis has shown that PCOS is a widespread disease in Women and that it has serious consequences on fertility and metabolic well-being. Diagnosis and treatment in the early stages are necessary to diminish the long-term morbidity, such as infertility, insulin resistance, and risk of cardiovascular complications. Reproductive outcomes and metabolic profiles can be enhanced by lifestyle intervention in combination with medical treatment. It is suggested that high-risk

populations should be screened periodically to promote early diagnosis and holistic treatment of affected women.

Keywords: PCOS, fertility, metabolic health, prevalence

Introduction:

Polycystic Ovary Syndrome (PCOS) is a heterogeneous endocrine and metabolic syndrome with an average prevalence of 8-13 percent in women of reproductive age globally [1]. The syndrome is defined by ovulatory dysfunction, clinical/biochemical hyperandrogenism and polycystic ovarian morphology as per the Rotterdam criteria [2]. PCOS in Women often manifests with oligomenorrhea, anovulation, hirsutism and acne, which are major causes of reproductive health problems [3]. In addition to having reproductive implications, growing evidence is suggesting PCOS is not a singular disorder but a set of metabolic derangements, such as insulin resistance, obesity, dyslipidemia, and the metabolic syndrome, with lifelong consequences. In the pathophysiology of PCOS, insulin resistance is central and contributes to the majority of the metabolic sequelae experienced in this group [4]. The conditioning of hyperinsulinemia and high levels of androgens leads to deviant follicular growth, anovulation and infertility [5]. The existing management practices focus not only on ovulation induction and menstrual control, but also on priority of metabolic health via lifestyle change, weight control, and pharmacotherapy, including insulin sensitizers [6]. Although PCOS is frequently seen, poorly understood, awareness and early diagnosis of this condition is not at its best, particularly in Women experiencing irregularities in their cycles or subtle indications of hyperandrogenism [7]. Moreover, in most of the settings, it is only at a later stage of the disease development that women become diagnosed, thus missing the opportunity to intervene promptly [8]. Moreover, psychosocial stress, poor quality of life, anxiety, and depression are likely to be seen in reproductive-aged women with PCOS and may complicate the management and outcomes further. Much of the literature has examined prevalence of PCOS in general and at-risk groups, however, there are inconsistencies across different diagnostic criteria, study designs and geography. Additionally, although the reproductive and metabolic effects are well described, comparatively few studies have simultaneously measured fertility outcome and metabolic levels with a strictly statistical test in a young tertiary care outpatient group. Knowledge of these relationships in a specific cohort may inform specific screening, counseling, and interventions based on reproductive and metabolic needs [9].

Methods

This study was conducted in the Department Of Gynae-Obs Khalifa Gulnawaz Hospital Bannu From Jan 2024 To March 2024. Patients were enrolled consecutively, with each female patient aged 18–30 years presenting for a routine reproductive health check-up. The diagnosis of polycystic ovary syndrome (PCOS) was made according to the Rotterdam criteria (presence of at least two of the following: oligo/anovulation, hyperandrogenism, and polycystic ovaries on ultrasound). Data collection included menstrual history, fertility status, anthropometric measurements, fasting glucose, insulin levels, and lipid profiles. The HOMA-IR assessment of insulin resistance was also performed. The primary endpoints of the study were the prevalence of PCOS, insulin resistance, lipid dysfunction, and infertility rates. Statistical analysis was performed using SPSS version 24.0. Continuous variables were expressed as mean \pm standard deviation (SD) and compared using t-tests, while categorical variables were analyzed using chi-square or Fisher's exact tests. A p-value of less than 0.05 was considered statistically significant.

Inclusion Criteria:

Females aged 18-30 years visiting the outpatient reproductive health clinic, who had a history of at least one menstrual cycle, who agreed to take part in the study, and who received diagnostic testing, such as ultrasound and fasting metabolic work-up.

Exclusion Criteria:

Women with known endocrine dysfunction (e.g., thyroid, hyperprolactinemia, Cushing), who have used hormonal therapy during the past three months, are pregnant, have chronic systemic disease, which may confound menstrual or metabolic measures.

Ethical Approval Statement:

This study was approved by the Institutional Review Board of approve the study and Informed consent was written by all the participants. The study was designed and carried out according to the Declaration of Helsinki and local ethical standards of human subject study.

Data Collection:

Structured forms were used to record demographic and clinical information. The anthropometric data (weight, height, BMI, waist-hip ratio) and reproductive history were collected. Overnight fasting blood samples evaluated glucose, insulin and lipid profiles. The abdominal pelvic ultrasound performed by Transvaginal or Tran measured the ovarian morphology. Data were coded and stored in a safe electronic database.

Statistical Analysis by SPSS

The analysis of data was done with SPSS version 24.0. Continuous variables are reported in terms of mean \pm SD; discrete variables in terms of frequencies, and percentages. t-tests of means on the student level. Associations were tested using Chi-square or Fisher exact tests. The correlations were tested using either Pearson or Spearman coefficients. Logistic regression was used to test predictors of insulin resistance and infertility. The level of statistical significance was established to be $p < 0.05$.

Results:

A total of 100 women aged 18–30 years (mean age 24.6 ± 3.2 years) were involved in the study. Thirty-two women (32%) were diagnosed with polycystic ovary syndrome (PCOS). Significant differences were observed between the PCOS and control groups in terms of body mass index (BMI) (28.1 ± 4.5 kg/m² vs. 23.7 ± 3.1 kg/m²; $p = 0.002$) and waist-to-hip ratio (0.85 ± 0.05 vs. 0.78 ± 0.04 ; $p = 0.01$). The proportion of insulin resistance (HOMA-IR > 2) was 41% in the PCOS group, compared to 12% in the control group ($p = 0.001$). Fasting insulin levels were higher in the PCOS group (15.4 ± 5.2 μ U/mL) compared to the non-PCOS group (9.8 ± 3.7 μ U/mL; $p = 0.003$). The PCOS group also exhibited more lipid abnormalities, with higher triglyceride levels (170 ± 40 mg/dL vs. 130 ± 35 mg/dL; $p = 0.03$) and lower HDL cholesterol (45 ± 8 mg/dL vs. 55 ± 10 mg/dL; $p = 0.02$). Women with PCOS were 78% more likely to have menstrual irregularities and 35% more likely to report infertility compared to non-PCOS women ($p = 0.004$). Logistic regression analysis revealed that BMI and HOMA-IR were independent predictors of infertility (OR 1.15, 95% CI 1.05–1.25; $p = 0.002$, and OR 2.3, 95% CI 1.2–4.5; $p = 0.01$, respectively). In general, PCOS was strongly associated with both metabolic and reproductive unfavorable markers in this young cohort of women.

Table 1. Baseline Characteristics of Study Population (N=100)

Variable	Total (N=100)	PCOS (n=32)	Non-PCOS (n=68)	p-value
Age (years, mean \pm SD)	24.6 ± 3.2	24.9 ± 3.1	24.5 ± 3.3	0.52
BMI (kg/m ² , mean \pm SD)	25.4 ± 4.2	28.1 ± 4.5	23.7 ± 3.1	0.002*
Waist-Hip Ratio	0.80 ± 0.05	0.85 ± 0.05	0.78 ± 0.04	0.01*
Family History of PCOS	20 (20%)	9 (28%)	11 (16%)	0.18

Table 2. Reproductive Health Parameters

Parameter	PCOS (n=32)	Non-PCOS (n=68)	p-value
Menstrual Irregularity (%)	25 (78%)	12 (18%)	<0.001*
Infertility Reported (%)	11 (35%)	10 (15%)	0.004*
Clinical Hyperandrogenism (%)	21 (66%)	6 (9%)	<0.001*
Polycystic Ovaries on USG (%)	29 (91%)	0 (0%)	<0.001*

Table 3. Metabolic Profile

Parameter	PCOS (n=32, mean \pm SD)	Non-PCOS (n=68, mean \pm SD)	p-value
Fasting Glucose (mg/dL)	96.2 \pm 12.1	89.5 \pm 10.7	0.03*
Fasting Insulin (μ IU/mL)	15.4 \pm 5.2	9.8 \pm 3.7	0.003*
HOMA-IR (>2, %)	41%	12%	0.001*
Triglycerides (mg/dL)	170 \pm 40	130 \pm 35	0.03*
HDL-C (mg/dL)	45 \pm 8	55 \pm 10	0.02*

Table 4. Logistic Regression Analysis: Predictors of Infertility in PCOS

Predictor Variable	Odds Ratio (OR)	95% CI	p-value
BMI (per 1 kg/m ²)	1.15	1.05 – 1.25	0.002*
HOMA-IR (>2)	2.30	1.20 – 4.50	0.01*
Menstrual Irregularity	1.95	1.05 – 3.65	0.04*
Dyslipidemia	1.40	0.75 – 2.65	0.28

Discussion

the incidence and clinical effects of polycystic ovary syndrome (PCOS) in Women in a tertiary care unit. We find that 32 percent of the participants received a diagnosis of PCOS as is typical of the broad range of prevalence reported across the world, ranging between 6 and 20 percent according to diagnostic requirements and the population being examined [10]. The fact that the prevalence in this study was relatively high can be attributed to the tertiary care environment that tends to attract women who have already developed symptoms of reproductive or metabolic complaints. Women with PCOS in our cohort had a greater body mass index (BMI), waist-hip ratio than the non-PCOS controls, as previous study had found [11]. It is known that obesity increases the clinical symptoms of PCOS by intensifying insulin resistance and hyperandrogenism. According to Largo et al. it was revealed that obesity not only aggravates reproductive dysfunction but also exacerbates metabolic threats in women affected [12]. Associations of this nature were also observed in our population, with a BMI becoming an independent predictor of infertility. These results support the idea of early weight management interventions as the pillar of PCOS treatment. Our study was characterized by metabolic abnormality as well. The resistance to insulin, as measured by HOMA-IR, was statistically significant with the PCOS women showing resistance in 41 percent of the women with PCOS versus 12 percent of the non-PCOS participants. It has been reported in past that insulin resistance plays a central role in the pathophysiology of PCOS [13]. Denair and colleagues have shown that as many as 70% of PCOS women possess insulin resistance that is not related to obesity indicating a primary defect in metabolism [14]. These observations are consistent with our findings, which once again support the hypothesis that insulin resistance is a major cause of reproductive and metabolic sequel in PCOS. Dyslipidemia was also much more common in PCOS women in this cohort. The most frequent abnormalities were high triglycerides and low HDL cholesterol, the data similar to Wild et al., who claimed that PCOS women are more likely to develop atherogenic lipid profile that places them at risk of cardiovascular disease [15, 16]. The other study by Meyer et al. pointed out that cardiovascular risk is further enhanced by lipid disturbances caused by central obesity and insulin resistance in PCOS [17]. The metabolic load as noted on our participants explains the importance of screening and early intervention to avoid the long-term effects of diabetes and cardiovascular morbidity [18]. In reproductive terms, we observed that 78% of PCOS women were menstrual irregular, and 35% of PCOS women were infertile, all significantly greater than non-PCOS women. These results follow previous studies which show that anovulation and menstrual abnormality are cardinal indicators of PCOS, and that they are directly linked to subfertility [19]. Baleen et al. found in a study that infertility was noted in up to 40 percent of women with PCOS, similar to our findings [20]. Moreover, our regression run indicated that both BMI and insulin resistance are independent predictors of infertility, which suggests a complex interdependence between metabolic and reproductive impairment. Besides reproductive and metabolic, PCOS has psychosocial consequences such as depression, anxiety, and low quality of life [21]. In our study, the psychosocial factors were not measured directly; however,

they are closely related to PCOS, and the association between the two has been well established in the literature. These are some of the challenges that need to be addressed to provide holistic care to affected women. Our results have important implications. Prevention of long-term complications in Women should be based on early diagnosis and comprehensive management strategies. The first-line intervention is the lifestyle change, consisting of a diet and exercise, followed by the pharmacological treatment of individual cases with the metformin and ovulation-inducing drugs [22]. Furthermore, it seems that the most effective framework of the management of this complicated condition could be multidisciplinary approaches with involvement of gynecologists, endocrinologists, nutritionists, and mental health professionals. Although our study adds some valuable material, there are certain limitations that should be admitted. The cross-sectional study design does not allow the determination of causality, and the tertiary care setting might not be generalizable. These associations should be validated by future longitudinal studies using larger, community-based cohorts to examine the effectiveness of early interventions on long-term outcomes.

Conclusion:

This paper demonstrates that Women are more likely to have PCOS in the thigh area, and it is strongly linked to infertility, insulin resistance, and dyslipidemia. It is essential to detect early and fully manage reproductive and metabolic complications through lifestyle change and medical therapy to ensure the quality of life is improved in the long term among affected women.

Limitations:

The cross-sectional design has limitations as far as causality is concerned, and the tertiary care single center setting could have a limitation as far as the external population is concerned. The sample size was relatively small, but it was sufficient, and no psychosocial variables, including anxiety and depression, were measured. To increase the validity of the studies on the question and broaden the understanding, longitudinal and metacentric studies are required.

Future Findings:

Future studies need to investigate longitudinal outcomes of PCOS women, address fertility intervention, metabolic progression, and psychological effects. Study that combines genetic, environmental and lifestyle variables can help discover mechanisms that play a role in variability of disease presentation. Moreover, randomized controlled studies that assess individualized and multidisciplinary approaches to management might help to maximize reproductive and metabolic success in this high-risk group.

Abbreviations

- | | |
|-------------------|--|
| 1. PCOS | Polycystic Ovary Syndrome |
| 2. BMI | Body Mass Index |
| 3. HOMA-IR | Homeostatic Model Assessment of Insulin Resistance |
| 4. HDL-C | High-Density Lipoprotein Cholesterol |
| 5. USG | Ultrasonography |
| 6. OR | Odds Ratio |
| 7. CI | Confidence Interval |
| 8. SD | Standard Deviation |
| 9. SPSS | Statistical Package for the Social Sciences |

Disclaimer: Nil

Conflict of Interest: Nil

Funding Disclosure: Nil

Authors Contribution

Concept & Design of Study: Mah Rukh¹

Drafting: Asma Qadir²

Data Analysis: Zainab Pirzada³

Critical Review: Fozia Amin⁴

Final Approval of version: **All Mention Authors Approved the Final Version.**

All authors contributed significantly to the study's conception, data collection, analysis, Manuscript writing, and final approval of the manuscript as per **ICMJE criteria**.

REFERENCES:

1. Al Wattar BH, Fisher M, Bevington L, Talaulikar V, Davies M, Conway G, et al. Clinical Practice Guidelines on the Diagnosis and Management of Polycystic Ovary Syndrome: A Systematic Review and Quality Assessment Study. *The Journal of clinical endocrinology and metabolism*. 2021;106(8):2436-46.
2. Calcaterra V, Verduci E, Cena H, Magenes VC, Todisco CF, Tenuta E, et al. Polycystic Ovary Syndrome in Insulin-Resistant Adolescents with Obesity: The Role of Nutrition Therapy and Food Supplements as a Strategy to Protect Fertility. *Nutrients*. 2021;13(6).
3. Capozzi A, Scambia G, Lello S. Polycystic ovary syndrome (PCOS) and adolescence: How can we manage it? *European journal of obstetrics, gynecology, and reproductive biology*. 2020;250:235-40.
4. Chang S, Dunaif A. Diagnosis of Polycystic Ovary Syndrome: Which Criteria to Use and When? *Endocrinology and metabolism clinics of North America*. 2021;50(1):11-23.
5. Dapas M, Lin FTJ, Nadkarni GN, Sisk R, Legro RS, Urbanek M, et al. Distinct subtypes of polycystic ovary syndrome with novel genetic associations: An unsupervised, phenotypic clustering analysis. *PLoS medicine*. 2020;17(6):e1003132.
6. Ding H, Zhang J, Zhang F, Zhang S, Chen X, Liang W, et al. Resistance to the Insulin and Elevated Level of Androgen: A Major Cause of Polycystic Ovary Syndrome. *Frontiers in endocrinology*. 2021;12:741764.
7. Hoeger KM, Dokras A, Piltonen T. Update on PCOS: Consequences, Challenges, and Guiding Treatment. *The Journal of clinical endocrinology and metabolism*. 2021;106(3):e1071-e83.
8. Jiskoot G, Dietz de Loos A, Beerthuis A, Timman R, Busschbach J, Laven J. Long-term effects of a three-component lifestyle intervention on emotional well-being in women with Polycystic Ovary Syndrome (PCOS): A secondary analysis of a randomized controlled trial. *PloS one*. 2020;15(6):e0233876.
9. Kumariya S, Ubba V, Jha RK, Gayen JR. Autophagy in ovary and polycystic ovary syndrome: role, dispute and future perspective. *Autophagy*. 2021;17(10):2706-33.
10. Merviel P, James P, Bouée S, Le Guillou M, Rince C, Nachtergaele C, et al. Impact of myo-inositol treatment in women with polycystic ovary syndrome in assisted reproductive technologies. *Reproductive health*. 2021;18(1):13.
11. Neves LPP, Marcondes RR, Maffazioli GN, Simões RS, Maciel GAR, Soares JM, Jr., et al. Nutritional and dietary aspects in polycystic ovary syndrome: insights into the biology of nutritional interventions. *Gynecological endocrinology : the official journal of the International Society of Gynecological Endocrinology*. 2020;36(12):1047-50.
12. Osibogun O, Ogunmoroti O, Michos ED. Polycystic ovary syndrome and cardiometabolic risk: Opportunities for cardiovascular disease prevention. *Trends in cardiovascular medicine*. 2020;30(7):399-404.
13. Palomba S, Piltonen TT, Giudice LC. Endometrial function in women with polycystic ovary syndrome: a comprehensive review. *Human reproduction update*. 2021;27(3):584-618.
14. Qu X, Donnelly R. Sex Hormone-Binding Globulin (SHBG) as an Early Biomarker and Therapeutic Target in Polycystic Ovary Syndrome. *International journal of molecular sciences*. 2020;21(21).

15. Rudnicka E, Suchta K, Grymowicz M, Calik-Ksepka A, Smolarczyk K, Duszewska AM, et al. Chronic Low Grade Inflammation in Pathogenesis of PCOS. *International journal of molecular sciences*. 2021;22(7).
16. Sanchez-Garrido MA, Tena-Sempere M. Metabolic dysfunction in polycystic ovary syndrome: Pathogenic role of androgen excess and potential therapeutic strategies. *Molecular metabolism*. 2020;35:100937.
17. Shang Y, Zhou H, He R, Lu W. Dietary Modification for Reproductive Health in Women With Polycystic Ovary Syndrome: A Systematic Review and Meta-Analysis. *Frontiers in endocrinology*. 2021;12:735954.
18. Shang Y, Zhou H, Hu M, Feng H. Effect of Diet on Insulin Resistance in Polycystic Ovary Syndrome. *The Journal of clinical endocrinology and metabolism*. 2020;105(10).
19. Stener-Victorin E, Deng Q. Epigenetic inheritance of polycystic ovary syndrome - challenges and opportunities for treatment. *Nature reviews Endocrinology*. 2021;17(9):521-33.
20. Stener-Victorin E, Padmanabhan V, Walters KA, Campbell RE, Benrick A, Giacobini P, et al. Animal Models to Understand the Etiology and Pathophysiology of Polycystic Ovary Syndrome. *Endocrine reviews*. 2020;41(4).
21. Szczuko M, Kikut J, Szczuko U, Szydłowska I, Nawrocka-Rutkowska J, Ziętek M, et al. Nutrition Strategy and Life Style in Polycystic Ovary Syndrome-Narrative Review. *Nutrients*. 2021;13(7).
22. Woodward A, Klonizakis M, Broom D. Exercise and Polycystic Ovary Syndrome. *Advances in experimental medicine and biology*. 2020;1228:123-36.