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Original Research Article

Correlation Between Polycystic Ovarian Syndrome Phenotypes and Metabolic Syndrome in Reproductive-Age Women

Anamika Ranjan[1], Anisha Buddhapriya[2], Sangeeta Sinha[3]

[1]Senior Resident, Department of Obs and Gynae, Patna Medical College and Hospital, Patna, Bihar, India

[2]Senior Resident, Department of Obs and Gynae, Patna Medical College and Hospital, Patna, Bihar, India

[3]Professor and HOD, Department of Obs and Gynae, Patna Medical College and Hospital, Patna, Bihar, India

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Corresponding Author: Anisha Buddhapriya

Conflict of interest: Nil

Abstract:

Background: Polycystic Ovary Syndrome is a common endocrine disorder in reproductive-age women and is associated with obesity, insulin resistance, dyslipidemia, and increased cardiovascular risk. Different PCOS phenotypes may carry varying metabolic burdens.

Objective: To assess the correlation between PCOS phenotypes and metabolic syndrome in reproductive-age women.

Methods: Over the course of ten months, a prospective observational study was carried out in the obstetrics and gynecology department of a tertiary care facility. Phenotypes A, B, C, and D were assigned to 100 women between the ages of 18 and 40 who had been diagnosed with PCOS following Rotterdam criteria. Anthropometry, blood pressure, fasting glucose, insulin, lipid profile, clinical examination, and hormonal assays were carried out. The modified NCEP ATP III criteria were used to diagnose metabolic syndrome. ANOVA, logistic regression, and the chi-square test were used in the statistical analysis.

Results: The most prevalent phenotype was A (34%), which was followed by C (26%), B (22%), and D (18%). 48% of people had metabolic syndrome overall. Phenotypes A (58.8%) and B (63.6%) had considerably greater rates of metabolic syndrome than phenotypes C (38.5%) and D (22.2%) ($p=0.003$). 62% of patients had insulin resistance, which was associated with increased waist circumference and BMI ($p<0.001$). The highest metabolic risk was seen in phenotypes with hyperandrogenism and anovulation.

Conclusion: PCOS phenotypes demonstrate heterogeneous metabolic risk. Classic phenotypes A and B are strongly associated with metabolic syndrome and warrant early metabolic screening and long-term follow-up.

Keywords: PCOS, heterogeneous, metabolic risk, screening, BMI.

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Introduction

One of the most prevalent endocrine conditions affecting women of reproductive age, polycystic ovary syndrome is a major contributor to anovulatory infertility, hyperandrogenism, and irregular menstruation. Although its prevalence varies based on community variables and diagnostic criteria, it is becoming more widely acknowledged as a significant global problem for women's health. PCOS is now recognized as a complex metabolic condition with significant long-term consequences that extends beyond reproductive symptoms [1]. Insulin resistance, central obesity, dyslipidemia, poor glucose tolerance, and an increased risk of type 2 diabetes mellitus are common in women with PCOS. These anomalies may come together to form metabolic syndrome, a disorder marked by higher triglycerides, decreased high-density lipoprotein cholesterol, hypertension, hyperglycemia, and abdominal obesity. The risk of cardiovascular disease and chronic metabolic morbidity in the future is significantly increased by metabolic syndrome [2].

Based on combinations of hyperandrogenism (HA), ovulatory dysfunction (OD), and polycystic ovarian morphology (PCOM), the Rotterdam criteria divide PCOS into four phenotypes. HA + OD + PCOM is included in phenotypic A; HA + OD is included in phenotype B; HA + PCOM is included in phenotype C; and OD + PCOM without hyperandrogenism is included in phenotype D. These phenotypes show differently in terms of reproduction and

biochemistry, and mounting data indicates that metabolic risk may also range considerably amongst them. Because both hyperandrogenism and ovulatory dysfunction are present, classic phenotypes A and B are frequently regarded as more severe. While persistent anovulation is often associated with obesity and metabolic disruption, hyperandrogenism may exacerbate visceral adiposity and insulin resistance. On the other hand, phenotype D can have a reduced cardiometabolic burden and lesser endocrine problems [3].

Data, however, is still uneven throughout hospital settings and ethnic groups. Rapid lifestyle changes, sedentary behaviors, and rising obesity may exacerbate the metabolic effects of PCOS in nations like India. Personalized screening, lifestyle counseling, and preventative care can be guided by early detection of high-risk phenotypes. Prospective research linking PCOS characteristics with metabolic syndrome in Indian women of reproductive age is still scarce, despite increased awareness.In order to assess the distribution of PCOS phenotypes and their association with metabolic syndrome among women visiting a tertiary care facility over a ten-month period, this prospective observational study was conducted. Assessing obesity, insulin resistance, and lipid abnormalities across phenotypes were secondary goals [4].

Methods

Study Design: Prospective observational study.

Study Duration: 10 months.

Setting: Department of Obstetrics and Gynaecology, tertiary care teaching hospital.

Sample Size: 100 reproductive-age women diagnosed with PCOS.

Inclusion Criteria

- Age 18–40 years
- Diagnosed with PCOS by Rotterdam criteria
- Willing to participate

Exclusion Criteria

- Pregnancy
- Thyroid dysfunction
- Hyperprolactinemia
- Cushing syndrome
- Congenital adrenal hyperplasia
- Use of hormonal medication in past 3 months

Assessments

- BMI, waist circumference
- Blood pressure
- Fasting glucose, insulin
- Lipid profile
- HOMA-IR
- Serum testosterone

Statistical Analysis: Chi-square test, ANOVA, logistic regression; significance at $p < 0.05$.

Results

Table 1: Baseline Characteristics

Variable	Value
Mean age (years)	25.9 ± 4.6
Mean BMI (kg/m²)	29.1 ± 5.2
Overweight/Obese	82 (82%)
Waist circumference >80 cm	74 (74%)
Family history of diabetes	36 (36%)

Table 2: Metabolic Syndrome Across Phenotypes

Phenotype	Total (n)	Metabolic Syndrome n (%)	p-value
A	34	20 (58.8)	
B	22	14 (63.6)	
C	26	10 (38.5)	
D	18	4 (22.2)	**0.003**

Table 3: Metabolic Parameters by Phenotype

Parameter	A	B	C	D	p-value
BMI (kg/m²)	30.6 ± 5.0	31.1 ± 5.4	27.8 ± 4.6	25.9 ± 4.2	0.008
HOMA-IR	3.8 ± 1.4	4.1 ± 1.6	3.0 ± 1.2	2.4 ± 1.0	<0.001
TG (mg/dL)	178 ± 42	184 ± 46	156 ± 38	142 ± 34	0.012

Table 4: Predictors of Metabolic Syndrome

Variable	Adjusted OR	95% CI	p-value
Phenotype A/B	3.42	1.46–8.02	0.004
BMI ≥30 kg/m²	4.18	1.72–10.14	0.001
HOMA-IR >2.5	3.76	1.51–9.33	0.005
Age >30 years	1.64	0.66–4.06	0.28

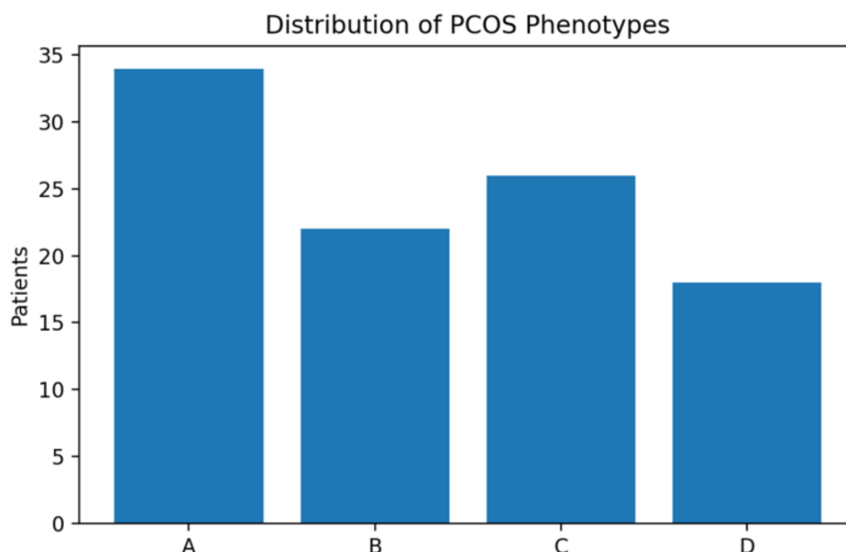

Figure 1: Distribution of PCOS phenotypes

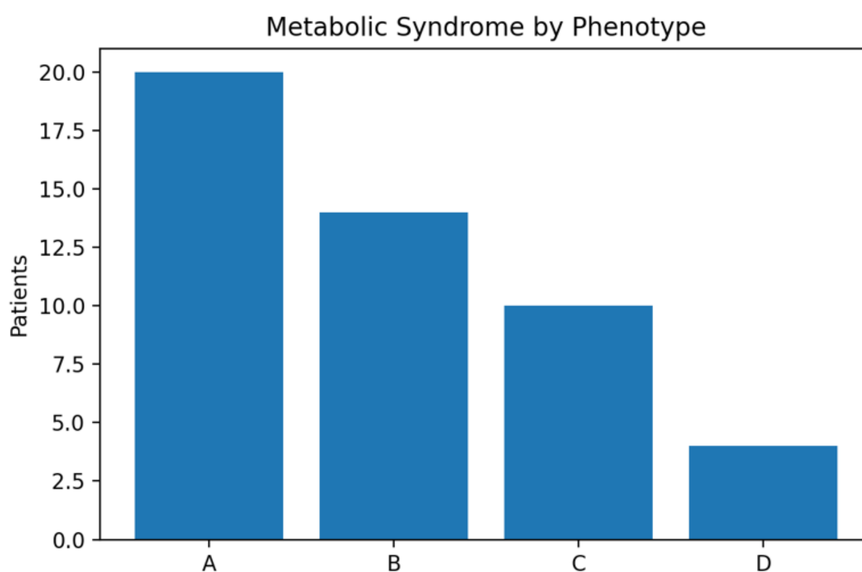

Figure 2: Metabolic syndrome by phenotype

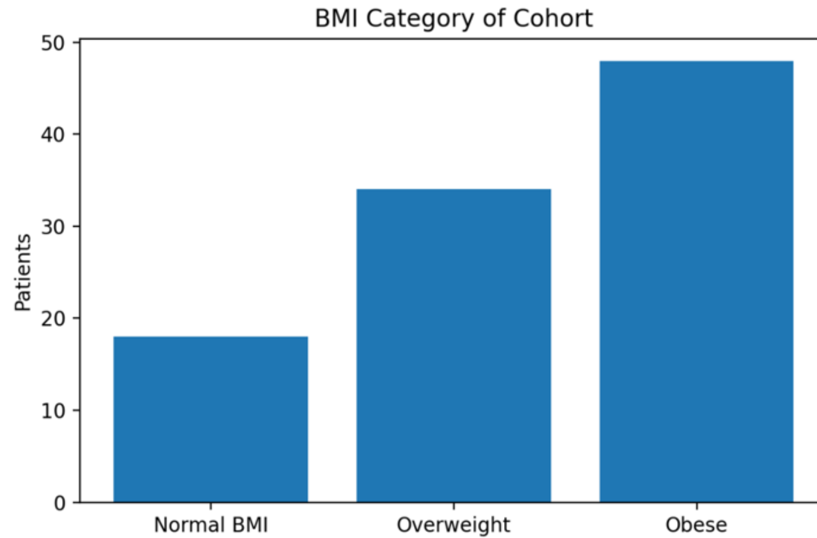

Figure 3: BMI category of cohort

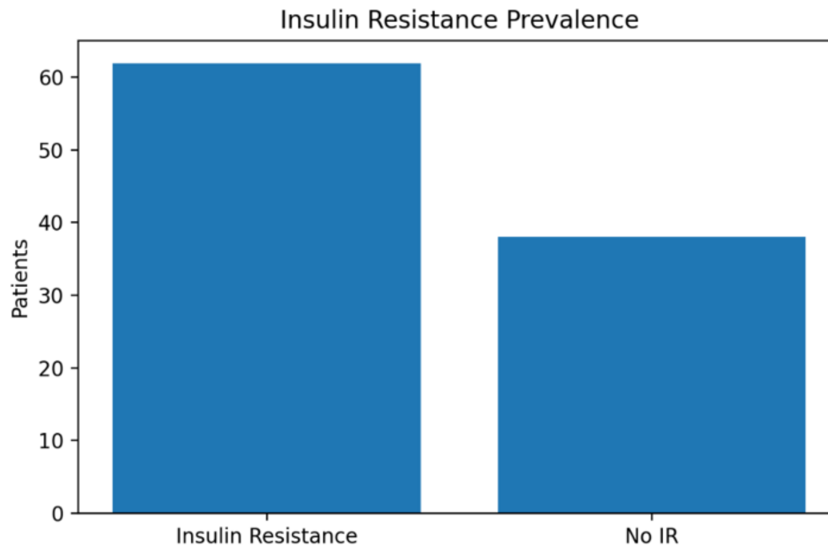

Figure 4: Insulin resistance prevalence

Discussion

This prospective observational study shows that different phenotypes of women with polycystic ovarian syndrome had different metabolic risks. Nearly half of the sample had metabolic syndrome, which was very prevalent overall but concentrated in the typical phenotypes A and B. These results lend credence to the idea that PCOS phenotypic classification has significant clinical significance outside of reproductive symptoms. The most prevalent presentation in our analysis was phenotypic A, which was followed by phenotypes C, B, and D. This pattern is similar to many groups receiving tertiary treatment, where women frequently exhibit more acute or symptomatic illness. Phenotype A is a complete manifestation of the condition, encompassing polycystic ovarian morphology, ovulatory failure, and hyperandrogenism [5].

Phenotype B likewise exhibits hyperandrogenism and anovulation, although not having ultrasonography polycystic appearance. The highest incidences of metabolic syndrome were seen in both phenotypes. This trend could be explained by a number of ways. Hepatic steatosis, insulin resistance, visceral obesity, and dyslipidemia are all linked to hyperandrogenism. Increased androgen levels may exacerbate metabolic signals and encourage the accumulation of belly fat. Obesity and ongoing insulin resistance frequently coincide with chronic anovulation, resulting in a vicious cycle of endocrine and metabolic dysfunction. Therefore, a

higher cardiometabolic burden is anticipated for women who display both hyperandrogenism and ovulatory disruption [6].

62% of participants had insulin resistance, which was closely linked to central obesity and a higher BMI. This is consistent with the current understanding that, although its severity varies among phenotypes, insulin resistance is a key pathophysiological characteristic of PCOS. Free androgen levels rise as a result of hyperinsulinemia's stimulation of ovarian androgen synthesis and suppression of sex hormone-binding globulin. Reproductive and metabolic disorders therefore reinforce one another. Metabolic syndrome was less common in phenotype D, which also had better metabolic indicators. This phenotype may be a milder endocrine form of PCOS with comparatively less metabolic disturbance because it does not exhibit hyperandrogenism. However, a minority of women with phenotype D still developed metabolic syndrome, suggesting that no phenotype is entirely risk-free. For every patient, routine screening is still crucial [7].

It is noteworthy that the cohort has a high rate of overweight and obesity. Over four-fifths of participants were overweight or obese, underscoring the significant impact that adiposity and lifestyle choices play in exacerbating PCOS symptoms. In addition to improving menstrual function, insulin sensitivity, and fertility outcomes, weight loss through calorie restriction, exercise, sleep optimization, and behavioral support continues to be the first-line treatment. Individualized care can benefit from these findings. It may be necessary to examine women with phenotypes A and B for nonalcoholic fatty liver disease, blood pressure, glucose intolerance, and dyslipidemia earlier and more frequently. In this subgroup, aggressive lifestyle modification and insulin-sensitizing medication may be especially helpful. On the other hand, patients with phenotypes C and D may exhibit more cosmetic or reproductive issues, but they still require routine metabolic evaluation [8].

The importance of phenotype-based counseling is further supported by the study. Many times, patients only think of PCOS as a menstruation or reproductive issue. Understanding long-term metabolic risk can enhance follow-up and adherence to lifestyle modifications. For young women who might otherwise overlook preventive options, integrating gynecologic and metabolic care is particularly important. The single-center design, small sample size, and absence of a non-PCOS control group are among its drawbacks. Socioeconomic factors, physical activity, and food consumption were not completely measured. It is impossible to establish causal linkages because this was an observational study. The study's duration did not include long-term cardiovascular consequences [9].

Notwithstanding these drawbacks, the work offers helpful prospective data that the metabolic burden of PCOS phenotypes varies significantly. Future multicenter longitudinal research should investigate whether phenotype-directed therapies can slow the development of cardiovascular disease and diabetes.

Conclusion

In reproductive-age women, this prospective observational study discovered a significant association between metabolic syndrome and polycystic ovarian syndrome characteristics. The load was considerably greater in classic phenotypes A and B, which contain both hyperandrogenism and ovulatory dysfunction, even though metabolic abnormalities were prevalent throughout the sample. Additionally, these phenotypes showed worse lipid profiles, increased insulin resistance, and higher BMI. In comparison to classic forms, phenotypes C and D often had less severe biochemical disruption, while phenotype D had the lowest metabolic risk. Nonetheless, metabolic syndrome persisted in some women with all phenotypes, suggesting that a baseline cardiometabolic assessment is necessary for all PCOS patients.

The results emphasize how crucial phenotype-based risk classification is to standard clinical practice. Early screening for obesity, hypertension, dyslipidemia, impaired glucose tolerance, and insulin resistance is recommended for women with phenotypes A and B. Structured lifestyle intervention and long-term monitoring should come next. Exercise and weight control continue to be key therapy strategies for all phenotypes. Physicians should understand that PCOS is a varied metabolic syndrome with varying long-term risks rather than only a reproductive issue. Phenotype-based personalized care may enhance results, maximize resource use, and lessen the burden of diabetes and cardiovascular disease in the future.

In conclusion, the metabolic expression of PCOS phenotypes varies greatly, and early preventive therapy for women of reproductive age can be guided by the identification of high-risk phenotypes.

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