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Research Article

The study of clinical, metabolic and hormonal profile of women presenting with polycystic ovary syndrome in relation to body mass index

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ABSTRACT

Background:
Polycystic ovary syndrome (PCOS) is a common endocrine disorder associated with reproductive, hormonal, and metabolic abnormalities. Although frequently linked with obesity, it also occurs in women with normal body mass index (BMI). **Methods:** This descriptive cross-sectional study included 80 women aged 20–38 years with infertility and diagnosed with PCOS based on Rotterdam criteria. Subjects were categorized into Normal group (BMI <23 kg/m²) and overweight/obese (BMI ≥23 kg/m²) groups. Clinical evaluation, anthropometric measurements, hormonal assays, lipid profile, and oral glucose tolerance test were performed. Statistical analysis was done using Student's *t*-test. **Results:** Forty percent of PCOS patients had normal BMI. Clinical features such as menstrual irregularities, hirsutism, and hormonal profiles were comparable in both groups. However, overweight/obese women showed significantly higher blood pressure, BMI, waist-to-hip ratio, insulin levels, and dyslipidemia. Insulin resistance was highly prevalent in both Normal group (80%) and overweight/obese (92.5%) groups. **Conclusion:** PCOS is present across all BMI categories with similar clinical and hormonal features. However, metabolic abnormalities are more pronounced in overweight/obese individuals. Insulin resistance is a key underlying feature irrespective of BMI, highlighting the need for routine metabolic screening in all PCOS patients.

Keywords:
PCOS, BMI, insulin resistance, dyslipidemia, infertility

INTRODUCTION

Polycystic ovary syndrome (PCOS), also known as hyperandrogenic anovulation or Stein–Leventhal syndrome, is a heterogeneous, multifactorial, and polygenic endocrine disorder affecting approximately 5–15% of women of reproductive age worldwide [1]. It is characterized by a combination of clinical and/or biochemical hyperandrogenism, menstrual irregularities ranging from amenorrhea to menorrhagia, and the

presence of polycystic ovaries on ultrasonography.

Several risk factors have been implicated in the development of PCOS, including genetic predisposition, sedentary lifestyle, and obesity. These factors contribute to an increased risk of long-term complications such as cardiovascular disease (CVD), insulin resistance, dyslipidemia, type 2 diabetes mellitus, hypertension, and certain malignancies [2].

Insulin resistance (IR) and compensatory hyperinsulinemia play a pivotal role in the pathophysiology of PCOS. They influence the hypothalamic–pituitary–ovarian axis, leading to increased ovarian androgen production. Interestingly, insulin resistance has also been reported in non-obese women with PCOS, suggesting that hyperinsulinemia can occur independently of obesity. Thus, the phenotypic expression of PCOS varies widely depending on body composition, androgen levels, and other metabolic factors [3].

Central obesity has been proposed as a key contributor to metabolic disturbances such as insulin resistance, dyslipidemia, and metabolic syndrome. However, the exact mechanism by which obesity influences the pathogenesis of PCOS remains unclear, and obesity is not included as a diagnostic criterion [4]. Additionally, biochemical hyperandrogenism has been observed to decline with advancing age, which has important implications for clinical diagnosis and management [5].

Dyslipidemia is commonly observed in women with PCOS and is typically characterized by elevated triglycerides and reduced high-density lipoprotein (HDL) cholesterol levels. However, variations in lipid profiles have been reported across different populations and geographical regions [6]. Given its high prevalence and significant reproductive, metabolic, and cardiovascular consequences, PCOS has been the focus of extensive research. Ethnicity, along with genetic and environmental factors, appears to significantly influence the clinical phenotype of PCOS [7].

Despite growing evidence, it remains unclear whether dyslipidemia and sex hormone profiles differ between women with PCOS and those with non-PCOS-related infertility. Furthermore, the relationship between sex hormones, anthropometric parameters, insulin resistance, and lipid profile in women with PCOS requires further investigation [4].

According to the consensus workshop jointly conducted by ESHRE/ASRM/FOGSI in Rotterdam in 2003, the diagnosis of PCOS is based on the presence of at least two of the following three criteria: (1) chronic anovulation or irregular menstrual cycles, (2) clinical and/or biochemical evidence of hyperandrogenism, and (3) polycystic ovaries on ultrasonography [8–10]. In contrast, the Androgen Excess and PCOS Society (AE-PCOS) criteria emphasize the presence of hyperandrogenism along with ovarian dysfunction and the exclusion of other related disorders.

Although overweight and obesity are frequently associated with PCOS, they are not essential for diagnosis. A considerable proportion of women with PCOS have a normal or even low body mass index (BMI). Recent studies suggest that insulin resistance is a fundamental feature in the development of PCOS and increases the risk of type 2 diabetes mellitus over time.

In this context, the present study aims to determine the proportion of PCOS patients who are lean (normal BMI) and to compare the clinical, hormonal, and metabolic profiles between lean and overweight/obese women with PCOS.

MATERIALS AND METHODS

This descriptive cross-sectional study was conducted in the Department of Gynaecology, SGT Medical College Hospital & Research Institute, Budhera, Gurugram, Haryana, India. A total of 80 consecutive women aged 20–38 years presenting with infertility over a period of three years and diagnosed with polycystic ovary syndrome (PCOS) according to the ESHRE/ASRM/FOGSI (Rotterdam) criteria [1] were included in the study after obtaining informed written consent.

Participants were categorized based on body mass index (BMI) into two groups: Normal group (BMI <23 kg/m²) and overweight/obese PCOS (BMI ≥23 kg/m²) [9]. A detailed clinical history was obtained, including menstrual history, personal habits, past medical and surgical history, family history, obstetric history, and prior treatment details.

A thorough physical examination was performed for all participants, including general, systemic, breast, and pelvic examinations. Clinical features of hyperandrogenism such as hirsutism, acne, acanthosis nigricans, clitoromegaly, and alopecia were assessed. Hirsutism was evaluated using the modified Ferriman–Gallwey scoring system, and a score >7 was considered indicative of hirsutism [10].

Anthropometric measurements were recorded using standard methods. Height and weight were measured to calculate BMI. Waist circumference was measured at the level of the umbilicus in a standing position without clothing, and hip circumference was measured at the level of the ischial tuberosities. The waist–hip ratio (WHR) was calculated, and a value ≥0.8 was considered abnormal.

Subjects with known thyroid disorders (including subclinical hypothyroidism), hypothalamic, pituitary, or adrenal disorders, neoplastic diseases, hepatic, renal, or cardiovascular diseases, and tuberculosis were excluded from the study.

Laboratory Investigations

All participants underwent a series of laboratory investigations, including complete blood count and erythrocyte sedimentation rate (ESR), fasting lipid profile, and hormonal profile. Hormonal assays were performed on day 2 or 3 of the menstrual cycle and included luteinizing hormone (LH), follicle-stimulating hormone (FSH), LH/FSH ratio, total testosterone, prolactin, and thyroid-stimulating hormone (TSH). Serum progesterone levels were measured during the mid-luteal phase (days 21–23 of the menstrual cycle).

Glucose metabolism was assessed using a 75 g oral glucose tolerance test (OGTT), during which blood glucose and serum insulin levels were measured. The results were interpreted according to the American Diabetes Association (ADA) 1997 criteria [11]. Insulin resistance was defined as fasting insulin levels >25 μU/mL or post-load insulin levels >41 μU/mL [12].

Lipid profile assessment was performed in accordance with the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III) guidelines [14]. Dyslipidemia was defined as the presence of any abnormal lipid parameter, with cutoff values of total cholesterol ≥200 mg/dL, triglycerides ≥150 mg/dL, and HDL cholesterol <50 mg/dL.

Ultrasonographic Evaluation

Transvaginal ultrasonography (TVS) of the pelvis was performed using a 7 MHz transvaginal probe on day 2 or 3 of the menstrual cycle or following withdrawal bleeding. Endometrial thickness was assessed, and ovarian morphology was evaluated. Polycystic ovaries were defined as the presence of 20 or more follicles measuring 2–9 mm in diameter and/or an ovarian

volume >10 cm^3, along with increased stromal echogenicity.

Statistical Analysis

Data were expressed as mean ± standard deviation (SD). Clinical, metabolic, and hormonal parameters were compared between lean and overweight/obese PCOS groups using the unpaired two-tailed Student's *t*-test. A *p*-value <0.05 was considered statistically significant. All statistical analyses were performed using SPSS software version 24.0.

OBSERVATIONS AND RESULTS

A total of 80 women with PCOS were included in the study and divided into two groups: Normal PCOS (BMI <23 kg/m^2; n = 40) and overweight/obese PCOS (BMI ≥23 kg/m^2; n = 40). Among the study population, 58% were overweight or obese, and 28% were obese (BMI ≥27 kg/m^2), while 40% had a normal BMI. The mean age was comparable between the groups (28.7 ± 3.5 years in normal vs. 29.2 ± 3.9 years in overweight; p = 0.548).

Clinical features such as hirsutism, irregular menstrual cycles, acne, acanthosis nigricans, and clitoromegaly were similar in both groups. However, systolic and diastolic blood pressures were significantly higher in the overweight/obese group. Hormonal profiles were also comparable, except for significantly higher serum testosterone in the overweight group and higher prolactin levels in the lean group.

Table 1: Anthropometric and Biochemical Assessment Among Groups

Characteristics	Normal PCOS (Mean ± SD / n %)	Obese PCOS (Mean ± SD / n %)	p-value
Age (years)	28.7 ± 3.5	29.2 ± 3.9	0.548
Systolic BP (mmHg)*	116.7 ± 9.6	121.3 ± 8.2	0.024
Diastolic BP (mmHg)*	74.6 ± 6.4	80.5 ± 6.4	0.0000092
Hirsutism	34 (85%)	36 (90%)	0.501
Hirsutism score	14.9 ± 2.5	15.6 ± 2.05	0.176
Irregular cycles	37 (92.5%)	39 (97.5%)	0.307
Acne	3 (7.5%)	8 (20%)	0.106
Polycystic ovaries (TVS)	40 (100%)	40 (100%)	—
Endometrial thickness >4 mm	3 (7.5%)	8 (20%)	0.106
Acanthosis nigricans	3 (7.5%)	6 (15%)	0.291
Clitoromegaly	1 (2.5%)	2 (5%)	0.558
Serum LH (mIU/mL)	16.2 ± 1.7	16.7 ± 1.8	0.205
LH/FSH ratio	2.6 ± 0.4	2.5 ± 0.5	0.326
Serum Testosterone (ng/dL)*	1.6 ± 0.1	1.7 ± 0.24	0.018
Serum Progesterone (ng/dL)	2.4 ± 0.6	2.18 ± 0.8	0.168
Serum FSH (mIU/mL)	4.4 ± 0.7	4.2 ± 0.7	0.204
Serum Prolactin (ng/dL)*	13.7 ± 1.5	12.1 ± 1.7	0.0000027
Serum TSH (mU/mL)	2.4 ± 0.37	2.34 ± 0.4	0.488

*

Statistically significant

Metabolic parameters showed significant differences between the two groups. Overweight/obese women had significantly higher BMI, waist-to-hip ratio, insulin levels, and dyslipidemia. Insulin resistance was highly prevalent in both groups but more common in overweight individuals

Table 2: Anthropometric and Metabolic Parameters in the Two Groups

Characteristics	Normal PCOS (Mean ± SD / n %)	Obese PCOS (Mean ± SD / n %)	p-value
BMI*	22.1 ± 1.9	28.7 ± 3.5	0.0000
Waist-to-hip ratio*	0.84 ± 0.15	1.06 ± 0.26	0.000018
Family history of diabetes	3 (7.5%)	13 (32.5%)	0.435
Deranged lipid profile*	5 (12.5%)	14 (35%)	0.018
Fasting plasma glucose (mg/dL)	76.6 ± 5.2	78.4 ± 4.98	0.118
2-hour postprandial glucose (mg/dL)*	89.05 ± 3.2	85.5 ± 5.3	0.001
Impaired GTT	2 (5%)	5 (12.5%)	0.238
Fasting insulin (mIU/mL)*	11.3 ± 0.95	19.7 ± 3.6	0.0000
2-hour postprandial insulin (mIU/mL)*	52.4 ± 5.1	94.3 ± 10.2	0.0000
Insulin resistance	32 (80%)	37 (92.5%)	0.106

*Statistically significant

Key Findings

- 40% of PCOS patients had normal BMI (lean PCOS).
- Clinical and hormonal features were similar in both groups.
- Overweight/obese PCOS showed:
  - Higher blood pressure
  - Increased dyslipidemia
  - Higher insulin levels
- Insulin resistance was present in both groups (80% **Normal** vs. 92.5% obese), highlighting its central role in PCOS irrespective of BMI.

DISCUSSION

Polycystic ovary syndrome (PCOS) is commonly associated with weight gain, obesity, and metabolic syndrome, including insulin resistance and dyslipidemia. The prevalence of obesity in women with PCOS is generally higher compared to healthy controls [15]. In the present study, we evaluated the anthropometric, hormonal, and metabolic variations among PCOS patients with respect to body mass index (BMI), with particular emphasis on insulin resistance and its association with other parameters.

Our findings are consistent with previous studies demonstrating that biochemical and hormonal profiles in PCOS vary according to BMI and age. Central obesity, reflected by an increased waist-to-hip ratio, was more prevalent in overweight/obese PCOS subjects, indicating a higher cardiometabolic risk. Anthropometric parameters showed worsening trends with increasing BMI, supporting earlier observations [5,15].

Although PCOS is often linked with obesity, a significant proportion (40%) of subjects

in the present study had a normal BMI, emphasizing that PCOS is not restricted to overweight individuals. Clinical manifestations such as menstrual irregularities, hirsutism, and acne, along with hormonal abnormalities including elevated LH, LH/FSH ratio, and testosterone levels, were observed in both lean and overweight/obese groups without significant differences. These findings suggest that the clinical and endocrine features of PCOS are largely independent of BMI.

Metabolic disturbances—including impaired glucose tolerance, dyslipidemia, elevated fasting and postprandial insulin levels, and insulin resistance—were present in both groups but were significantly more pronounced in overweight/obese PCOS patients. Similar observations have been reported in earlier studies [12–14]. The prevalence of lipid abnormalities in PCOS has been reported to be as high as 70%, further supporting our findings [16].

A key observation of this study is that insulin resistance is highly prevalent in PCOS irrespective of BMI, being present in 80% of lean and 92.5% of overweight/obese subjects. This highlights insulin resistance as a fundamental defect in the pathogenesis of PCOS rather than merely a consequence of obesity. Hyperinsulinemia plays a central role by influencing multiple pathways:

- At the hypothalamic–pituitary level, it enhances LH secretion, contributing to anovulation.
- At the hepatic level, it reduces the production of sex hormone-binding globulin (SHBG), leading to increased free androgen levels.
- At the ovarian level, it stimulates androgen production, resulting in clinical features such as hirsutism and acne.

Furthermore, insulin resistance contributes to the development of dyslipidemia by increasing triglyceride levels and reducing high-density lipoprotein (HDL) cholesterol. These changes predispose individuals to atherosclerosis, hypertension, and long-term cardiovascular disease [20,21].

Obesity further exacerbates these metabolic abnormalities. In the present study, overweight/obese PCOS patients demonstrated a higher prevalence of deranged lipid profile, impaired glucose tolerance, elevated postprandial insulin levels, and acanthosis nigricans—an established marker of insulin resistance. A positive family history of diabetes was also more common in this group, suggesting a possible genetic predisposition. These findings are in agreement with recent studies identifying obesity as a significant predictor of metabolic syndrome in PCOS [22].

The glucose tolerance test (GTT) was found to be a more sensitive indicator of metabolic dysfunction compared to isolated fasting or postprandial glucose measurements, supporting its routine use in the evaluation of PCOS patients [13]. Overall, the findings suggest that while obesity amplifies metabolic risk, insulin resistance is an intrinsic feature of PCOS, likely influenced by genetic and environmental factors.

Conclusion

A significant proportion (40%) of women with PCOS had normal BMI, yet showed clinical and hormonal features similar to overweight/obese patients. Insulin resistance was highly prevalent in both groups, indicating it as a key underlying factor in PCOS. However, metabolic abnormalities such as dyslipidemia, impaired glucose tolerance, and higher insulin levels were more common in overweight/obese PCOS. Regular metabolic screening is essential in all PCOS patients, irrespective of BMI.

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