Section : OBG

Original Research Article

CARDIOMETABOLIC RISK COMPARISON IN PCOS AND NORMAL WOMEN

Geethanjali G[1], Divyatha R[2], Manisha Sharma[3], Harsh Vardhan Singh[4]

[1]*Assistant Professor, CIMS, Chikmagalur, Karnataka, India*
[2]*Senior Specialist, Department of OBG, K.R. Nagar, Karnataka, India*
[3]*Senior Consultant, Department of OBG, Hindu Rao Hospital, Delhi, India*
[4]*Senior Biochemist, Department of Biochemistry Hindu Rao Hospital, Delhi, India*

Received *: 22/08/2025*
Received in revised form *: 07/10/2025*
Accepted *: 28/10/2025*

Corresponding Author:
Dr. Divyatha R
Senior Specialist, Department of OBG, K.R. Nagar, Karnataka, India.
Email: divyathakims.kims@gmail.com

DOI: 10.70034/ijmedph.2025.4.132

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2025; 15 (4); 740-743

ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) is a common endocrine disorder characterized by hyperandrogenism, insulin resistance, and metabolic derangements that predispose women to cardiovascular disease. The aim is to compare endocrinological and metabolic parameters between women with PCOS and normally ovulating controls, highlighting early markers of cardiometabolic risk.

Materials and Methods: This hospital-based observational study included 70 women (35 PCOS and 35 controls) aged 18–35 years. Diagnosis of PCOS was based on Rotterdam criteria. Anthropometric, hormonal, and biochemical parameters were assessed including LH, FSH, LH/FSH ratio, SHBG, testosterone, free androgen index (FAI), fasting plasma glucose, fasting insulin, HOMA-IR, and triglycerides. Statistical analysis was done using SPSS version 24.0 with Mann–Whitney U test.

Results: Women with PCOS exhibited significantly higher BMI, systolic and diastolic blood pressure, fasting plasma glucose, fasting insulin, HOMA-IR, and triglyceride levels compared to controls. SHBG levels were markedly lower, while testosterone and FAI were elevated. These findings demonstrate a concurrent endocrine and metabolic dysregulation, predisposing PCOS patients to early cardiometabolic risk.

Conclusion: PCOS is associated with significant hormonal and metabolic disturbances including insulin resistance, dyslipidemia, and reduced SHBG levels. Early screening for these risk factors is essential to prevent long-term cardiovascular complications.

Keywords: PCOS, SHBG, Insulin resistance, HOMA-IR, Dyslipidemia, Cardiometabolic risk.

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is one of the most prevalent endocrine disorders among reproductive-aged women, with an estimated prevalence of 10% according to Rotterdam criteria in India.[1] It is characterized by ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology, leading to reproductive, metabolic, and cardiovascular complications.

Women with PCOS often exhibit insulin resistance, compensatory hyperinsulinemia, and dyslipidemia even in the absence of obesity.[2] Insulin resistance plays a central role, enhancing ovarian androgen production and reducing hepatic synthesis of sex hormone-binding globulin (SHBG), thus increasing bioavailable testosterone.[3]

QThis constellation of abnormalities predisposes women with PCOS to hypertension, type 2 diabetes, and cardiovascular disease.[4] The current study evaluates endocrinological and metabolic parameters to assess cardiometabolic risk among women with PCOS compared to age-matched controls.

MATERIALS AND METHODS

Study Design and Participants: This cross-sectional observational study was conducted in the Department of Obstetrics and Gynaecology, Hindu Rao Hospital, Delhi, after ethical approval (Ref No. HRH/2017/6972). Seventy women aged 18–35 years were included—35 diagnosed with PCOS (based on Rotterdam ESHRE/ASRM criteria) and 35 normally ovulating women with regular cycles (21–35 days).

Inclusion Criteria

- Women aged 18–35 years
- Diagnosed with PCOS according to Rotterdam criteria (≥2 of: oligo/anovulation, hyperandrogenism, polycystic ovaries on ultrasound)

Exclusion Criteria

- Thyroid disorders, Cushing's syndrome, congenital adrenal hyperplasia
- Diabetes mellitus or hypertension
- Current use of hormonal therapy or lipid-altering medication

Measurements: Height, weight, and blood pressure were measured using standard protocols. BMI was calculated as weight/height² (kg/m²). Blood samples were collected on day 3 of the menstrual cycle for biochemical analysis.

Serum LH, FSH, testosterone, DHEA-S, and SHBG were measured by electro-chemiluminescence immunoassay (ECLIA, Roche Cobas e411). Free Androgen Index (FAI) was derived as (total testosterone / SHBG) × 100. Fasting plasma glucose and lipid profile were analyzed on a COBAS INTEGRA 400 Plus autoanalyzer.

Insulin resistance was assessed using the Homeostatic Model Assessment formula:

HOMA-IR = (Fasting insulin × Fasting glucose) / 22.5

Statistical Analysis: Data were expressed as mean ± SD. Group comparisons were made using Mann–Whitney U test with significance at $p < 0.05$. Statistical analysis was done using IBM SPSS version 24.0.

RESULTS

A total of 70 women were included in the study, comprising 35 women diagnosed with Polycystic Ovary Syndrome (PCOS) and 35 normally ovulating controls. The mean age of women in both groups was comparable (23.5 ± 2.6 years in PCOS vs. 24.0 ± 2.4 years in controls; $p > 0.05$), confirming that both groups were age-matched. The detailed anthropometric, endocrine, and metabolic profiles of both groups are summarized in [Table 1].

Anthropometric Parameters: The mean Body Mass Index (BMI) of women with PCOS (25.9 ± 2.1 kg/m²) was significantly higher compared to controls (23.6 ± 1.8 kg/m²; $p < 0.05$). Elevated BMI among PCOS participants reflects the well-documented association between obesity and PCOS pathogenesis, possibly due to altered adipokine signaling and chronic low-grade inflammation that exacerbate insulin resistance and hyperandrogenism.

Systolic and diastolic blood pressures were also significantly higher among PCOS women (SBP = 118.1 ± 6.2 mmHg, DBP = 78.8 ± 4.5 mmHg) than controls (SBP = 113.5 ± 5.4 mmHg, DBP = 73.9 ± 3.8 mmHg; both $p < 0.05$). Although the absolute values remained within normal limits, the observed upward trend indicates early vascular changes and possible endothelial dysfunction associated with metabolic syndrome.

Gonadotropin Profile: Serum luteinizing hormone (LH) levels were markedly elevated in PCOS (14.9 ± 5.8 IU/L) compared with controls (7.1 ± 2.0 IU/L; $p < 0.01$), while follicle-stimulating hormone (FSH) levels were significantly lower (7.2 ± 1.6 IU/L vs. 9.3 ± 3.1 IU/L; $p < 0.05$). Consequently, the LH/FSH ratio was nearly tripled in PCOS (2.07 ± 1.11) versus controls (0.76 ± 0.24; $p < 0.01$).

This elevated LH/FSH ratio reflects ovarian theca cell hyperactivity and impaired follicular maturation, both characteristic features of PCOS. The gonadotropin imbalance promotes excessive androgen synthesis and anovulation.

Androgenic and SHBG Profile: Serum Sex Hormone Binding Globulin (SHBG) levels were significantly lower in the PCOS group (45.6 ± 13.4 nmol/L) compared with controls (70.2 ± 14.7 nmol/L; $p < 0.01$). The reduction in SHBG among PCOS women is attributed to hyperinsulinemia, as insulin inhibits hepatic SHBG synthesis.

Conversely, total testosterone levels were significantly elevated in PCOS (1.42 ± 0.39 ng/mL) relative to controls (0.74 ± 0.23 ng/mL; $p < 0.001$). The Free Androgen Index (FAI), which reflects the biologically active fraction of circulating testosterone, was more than doubled in the PCOS group (10.6 ± 3.4%) compared to controls (4.5 ± 1.6%; $p < 0.001$).

This clear biochemical hyperandrogenism supports the clinical picture of hirsutism and acne often observed in PCOS. The inverse relationship between SHBG and FAI further demonstrates insulin-mediated suppression of SHBG and resultant androgen excess.

Glucose–Insulin Dynamics: Fasting plasma glucose levels were significantly higher in PCOS women (91.7 ± 8.5 mg/dL) compared to controls (84.9 ± 7.2 mg/dL; $p < 0.01$). Although still within the normal fasting range, the increase suggests early disturbances in glucose tolerance.

Fasting serum insulin concentrations were markedly elevated in PCOS (15.8 ± 5.1 µIU/mL) versus controls (8.9 ± 2.8 µIU/mL; $p < 0.001$). Correspondingly, the mean HOMA-IR index—a measure of insulin resistance—was significantly higher among PCOS participants (3.62 ± 1.02) compared with controls (1.51 ± 0.59; $p < 0.001$).

These findings confirm that insulin resistance and compensatory hyperinsulinemia are key metabolic features of PCOS. Such derangements contribute to

both androgen excess and long-term cardiometabolic risk.

Lipid Profile: Serum triglyceride levels were significantly elevated in PCOS women (149.3 ± 44.8 mg/dL) compared with controls (118.7 ± 25.9 mg/dL; $p < 0.01$). Hypertriglyceridemia in PCOS is largely insulin-driven, as excess insulin promotes hepatic triglyceride synthesis and inhibits lipolysis. Dyslipidemia in these patients thus represents a precursor state for atherosclerosis and cardiovascular disease.

Table 1: Comparison of anthropometric, endocrinological, and metabolic parameters in PCOS and control groups (mean ± SD)

Parameter	PCOS (n = 35)	Controls (n = 35)	p-value*
Age (years)	23.5 ± 2.6	24.0 ± 2.4	NS
BMI (kg/m²)	25.9 ± 2.1	23.6 ± 1.8	< 0.05
SBP (mmHg)	118.1 ± 6.2	113.5 ± 5.4	< 0.05
DBP (mmHg)	78.8 ± 4.5	73.9 ± 3.8	< 0.05
LH (IU/L)	14.9 ± 5.8	7.1 ± 2.0	< 0.01
FSH (IU/L)	7.2 ± 1.6	9.3 ± 3.1	< 0.05
LH/FSH ratio	2.07 ± 1.11	0.76 ± 0.24	< 0.01
SHBG (nmol/L)	45.6 ± 13.4	70.2 ± 14.7	< 0.01
Testosterone (ng/mL)	1.42 ± 0.39	0.74 ± 0.23	< 0.001
Free Androgen Index (%)	10.6 ± 3.4	4.5 ± 1.6	< 0.001
Fasting Plasma Glucose (mg/dL)	91.7 ± 8.5	84.9 ± 7.2	< 0.01
Fasting Insulin (µIU/mL)	15.8 ± 5.1	8.9 ± 2.8	< 0.001
HOMA-IR	3.62 ± 1.02	1.51 ± 0.59	< 0.001
Triglycerides (mg/dL)	149.3 ± 44.8	118.7 ± 25.9	< 0.01

Statistical significance based on Mann–Whitney U test. NS = Not Significant.

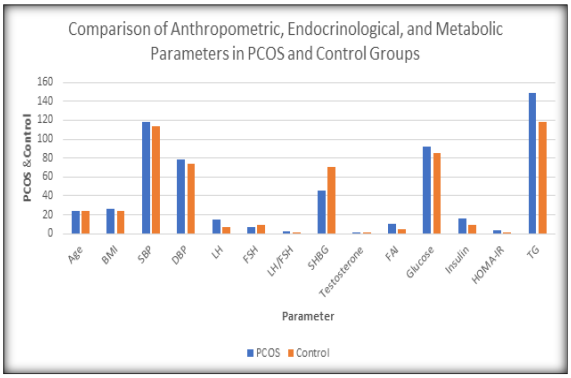

Figure 1: Comparison of Anthropometric, Endocrinological, and Metabolic Parameters in PCOS and Control Groups

Findings: PCOS women showed significantly higher BMI, systolic and diastolic blood pressure, fasting glucose, insulin, HOMA-IR, and triglyceride levels compared to controls. SHBG was significantly lower, whereas testosterone and FAI were elevated. These findings collectively indicate the presence of insulin resistance, compensatory hyperinsulinemia, and biochemical hyperandrogenism in PCOS.

DISCUSSION

The present study confirms that women with PCOS exhibit both endocrinological and metabolic abnormalities contributing to early cardiometabolic risk. The elevated BMI and blood pressure observed are consistent with previous studies by Wu et al,[2] and Mellembakken et al,[3] who found a 62% higher prevalence of hypertension among PCOS women.

The raised LH/FSH ratio and increased testosterone levels in our cohort reflect ovarian dysfunction and hyperandrogenism, hallmark features of PCOS. Reduced SHBG levels further augment free androgen availability, intensifying clinical manifestations such as hirsutism and acne.[4-6]

Insulin resistance was evident from elevated fasting insulin and HOMA-IR values. Similar findings were reported by Mishra et al,[7] showing higher insulin resistance in PCOS compared with controls. Hyperinsulinemia suppresses SHBG synthesis, creating a vicious cycle of increased free androgens and worsening metabolic profile.[8]

The significant elevation in triglycerides aligns with findings of Talbott et al,[6] and Behboudi-Gandevani et al,[5] who documented higher dyslipidemia and cardiovascular risk in PCOS women.

Thus, PCOS should not be viewed as a purely reproductive disorder but as a complex metabolic syndrome requiring early lifestyle modification and metabolic screening to reduce long-term cardiovascular complications.

CONCLUSION

PCOS women demonstrated significant increases in BMI, blood pressure, fasting glucose, insulin, HOMA-IR, and triglyceride levels, along with reduced SHBG and elevated testosterone and FAI. These findings underscore the coexistence of metabolic and endocrine derangements, increasing future risk of cardiovascular and diabetic complications.

Early identification and management through lifestyle intervention and metabolic monitoring are crucial to prevent progression to overt cardiometabolic disease.

REFERENCES

1. Bharali MD, Rajendran R, Goswami J, Singal K, Rajendran V. Prevalence of Polycystic Ovarian Syndrome in India: A Systematic Review and Meta-Analysis. Cureus. 2022 Dec 9;14(12):e32351. doi: 10.7759/cureus.32351. PMID: 36628015; PMCID: PMC9826643.
2. Wu CH, Chiu LT, Chang YJ, Lee CI, Lee MS, Lee TH, Wei JC. Hypertension risk in young women with polycystic ovary syndrome: a nationwide population-based cohort study. Front Med. 2020;7:574651.
3. Mellembakken JR, Mahmoudan A, Mørkrid L, Sundström-Poromaa I, Morin-Papunen L, Tapanainen JS, et al. Higher blood pressure in normal-weight women with PCOS compared to controls. Endocr Connect. 2021;10(2):154–63.
4. Markus AT, Seifert-Klauss V, Luppa PB. The biomarker sex hormone-binding globulin – from established applications to emerging trends in clinical medicine. Best Pract Res Clin Endocrinol Metab. 2015;29(5):749–60.
5. Behboudi-Gandevani S, Tehrani FR, Hosseinpanah F, Khalili D, Cheraghi L, Kazemijaliseh H, et al. Cardiometabolic risks in polycystic ovary syndrome: long-term population-based follow-up study. Fertil Steril. 2018;110(7):1377–86.
6. Talbott EO, Zborowski JV, Rager JR, Boudreaux MY, Edmundowicz DA, Guzick DS. Evidence for an association between metabolic cardiovascular syndrome and coronary and aortic calcification among women with polycystic ovary syndrome. J Clin Endocrinol Metab. 2004;89(11):5454–61.
7. Mishra S, Das AK, Das S. Hypovitaminosis D and associated cardiometabolic risk in women with PCOS. J Clin Diagn Res. 2016;10(5):BC01–4.
8. Stepto NK, Cassar S, Joham AE, Hutchison SK, Harrison CL, Goldstein RF, Teede HJ. Women with polycystic ovary syndrome have intrinsic insulin resistance on euglycaemic–hyperinsulinaemic clamp. Hum Reprod. 2013;28(3):777–84.