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Original article

DOI: https://doi.org/10.3329/jacedb.v5i1.84774

Journal of Association of Clinical Endocrinologist and Diabetologist of Bangladesh
January 2026, Vol. 5, No. 1, pp. 12-20

ISSN (Online): 2959-8176
ISSN (Print): 2958-0307

Dyslipidemia varies among the metabolic phenotypes of polycystic ovary syndrome

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Abstract

Background: The lipid profile among women with polycystic ovary syndrome (PCOS) is influenced by both insulin resistance and hyperandrogenism. The association of lipid profiles in metabolic phenotype-predominant women with PCOS was not adequately evaluated.

Objective: To assess the association of lipid profiles among metabolic phenotypes [body mass index (BMI) and insulin resistance (IR)] between women with and without PCOS.

Methods: This case-control study included 287 reproductive-aged females with PCOS and 190 age-matched controls conveniently sampled from the PCOS clinic, Department of Endocrinology of a University hospital, between September 2020 and August 2022. The fasting glucose, insulin, and lipid profiles were analyzed using glucose oxidase, chemiluminescence immunoassay, and glycerol phosphate dehydrogenase peroxidase methods. LDL cholesterol was calculated from Friedwald's formula or measured directly. The National Cholesterol Education Program's risk categories for different lipid fractions in Adult Treatment Panel III were used. Participants were divided into lean (<23 kg/m^2), overweight (23 – 27.4 kg/m^2), and obese (≥27.5 kg/m^2) by BMI, and IR by homeostasis model assessment of insulin resistance (HOMA-IR) ≥2.6.

Results: Approximately 70.7% of women with PCOS had at least one abnormality in their lipid profile. Among all lipid fraction classifications, only the high-risk LDL-cholesterol category showed a significant association with PCOS after adjustment for BMI and HOMA-IR status [OR=9.2, p=0.041]. Different lipid fractions differed between the study groups only in the lean-overweight and insulin-sensitive groups. However, none of the diagnostic features, including phenotypes, had significant associations with lipid fraction categories.

Conclusions: Only LDL cholesterol had an independent association with PCOS. An association between lipid fractions and PCOS was observed among women without obesity or IR. *[J Assoc Clin Endocrinol Diabetol Bangladesh,* January 2026; 5(1): *12-20]*

Keywords: Polycystic ovary syndrome, Lipid profile, Cholesterol, Dyslipidemia, Insulin resistance, Obesity

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Introduction

Polycystic ovary syndrome (PCOS) is a common chronic endocrine and metabolic condition with heterogeneous presentation. Reproductive and cutaneous features begin to develop after menarche, and metabolic features become more prominent over time. After menopause, the cardiometabolic features dominate and remain for the rest of their life.[1] PCOS is now an established risk factor for cardiovascular morbidity and mortality.[2] Although the overall risk is low in premenopausal women with PCOS, they have a higher prevalence of several cardiometabolic risk factors, including general and central obesity, abnormal glycemic status, dyslipidemia, metabolic

dysfunction-associated steatotic liver disease, metabolic syndrome, obstructive sleep apnea, insulin resistance, etc., during the reproductive years.[2] Among them, dyslipidemia has the highest population-attributable risk (47%) among women for myocardial infarction.[3] Dyslipidemia is associated with inflammation, oxidative stress, atherosclerosis, and other metabolic dysfunctions that exacerbate cardiovascular complications.[4] Hence, the current International Evidence-based Guideline, 2023 for PCOS, recommended testing for lipid profile irrespective of obesity among all women with PCOS at diagnosis, with other cardiometabolic risk assessments.[2]

Genetic, environmental, and lifestyle factors are related to abnormal lipid metabolism. Dyslipidemia is common among all 1st - degree relatives of women with PCOS.[5] Dyslipidemia in PCOS may be due to secondary causes such as physical inactivity, obesity, dysglycemia, hypothyroidism, use of hormonal contraceptives, etc. However, hyperandrogenism is another cardinal feature of PCOS that may also play a role in lipid metabolism through interactions with apolipoprotein E, endothelial lipase, hepatic lipase, and lipoprotein lipase.[6] Dyslipidemia may be related to hyperandrogenemia by several mechanisms, and it may cause mitochondrial dysfunction with oxidative stress.[7, 8] The beneficial effects of statins on androgens, inflammatory markers, and metabolic profiles, including lipids, were also demonstrated in several meta-analyses among women with PCOS.[9] The combined effects of hyperandrogenism and insulin resistance on lipid profiles among women with PCOS might be different from those in the general population, which needs to be evaluated.[10]

Ethnicity plays a significant role in the expression of PCOS features. Despite having a lower body mass index (BMI), women with PCOS from South-Asian backgrounds have higher visceral fat, hirsutism, metabolic features, and lower sex-hormone-binding globulin (SHBG).[11] Besides, diagnostic criteria may impact lipid profile.[12] Although the current guideline has adopted the Rotterdam criteria, definitions of each diagnostic component and the approach have changed significantly. PCOS is a heterogeneous condition where metabolic status plays a significant role in the expression of reproductive features.[6] We hypothesized that the interaction of metabolic and androgenic effects might affect lipid profiles differently among women with PCOS in comparison to the control group. This study aimed to compare the lipid fractions between PCOS and healthy controls based on metabolic phenotype defined by BMI and insulin resistance (IR). We also evaluated the association of lipid profiles with women with PCOS and their characteristics, diagnosed based on recent guidelines.

Methods

This case-control study was conducted among 287 reproductive-aged females and 190 age-matched controls who were conveniently enrolled from the PCOS clinic, Department of Endocrinology of Bangladesh Medical University, Dhaka, between September 2020 and August 2022. Ethical approval (R/N no: 3582; BSMMU/2021/7642, date: 24/08/2021) and informed consent were obtained from the University's Institutional Review Board (IRB) and each participant, respectively.

We diagnosed women with PCOS based on the International Evidence-based Guideline, 2018 [any 2 for adults: irregular menstrual cycle, hyperandrogenism, and polycystic ovarian morphology (PCOM), 1st two for adolescents].[13] Significant hirsutism was considered with a modified Ferriman-Gallwey score ≥6, hyperandrogenemia with a free androgen index (FAI) ≥ 5%, and PCOM by any ovarian volume ≥10 mL, irrespective of the route of ultrasonography.[13,14] Women without any of the diagnostic features were considered for controls. Additionally, having similar endocrinopathies, a history of taking hormonal contraceptives, insulin sensitizers, ovulation-inducing drugs, anti-androgen, anti-obesity, anti-lipid, valproate, etc., within three months of enrollment, any decompensated systemic diseases, current/ past history of taking alcohol, smoking, current pregnancy, and lactating mother were excluded from both groups.

Following relevant history-taking, physical examinations were performed, and fasting blood was drawn in the follicular phase of the menstrual cycle to measure glucose, lipid profile, total testosterone (TT), SHBG, and insulin. Glucose, lipids, and all hormones, including SHBG, were analyzed by glucose oxidase, glycerol phosphate dehydrogenase peroxidase, and chemiluminescent microparticle immunoassay methods, respectively. Low-density lipoprotein (LDL) was calculated from Friedwald's formulae or directly if triglyceride (TG) levels exceeded 400 mg/dL.[15] Non-high-density lipoprotein (non-HDL) cholesterol was calculated by subtracting HDL cholesterol from total cholesterol (TC). Atherogenic dyslipidemia was considered if both criteria were fulfilled: TG ≥150 mg/dL and HDL cholesterol <50 mg/dL. All the definitions of different risk categories of lipid fractions were taken from the definitions of the National

Cholesterol Education Program (NCEP), Adult Treatment Panel (ATP) III.[16] Ultrasonography was performed in the follicular phase of the menstrual cycle. A BMI of 23 kg/m^2 and 27.5 kg/m^2, a homeostasis model assessment of IR (HOMA-IR) of 2.6, were considered as cut-offs for defining BMI status (lean vs. overweight vs. obese), and HOMA-IR status (insulin sensitive vs. insulin resistant), respectively.[17,18]

SPSS software (version 25.0) was used to analyze the data. Data were expressed as frequency (%) or median (interquartile range, IQR), depending on their type. Pearson's chi-square test was used to analyze the association between two categorical variables. For numerical variables, association with two-group categorical variables was tested using the Mann-Whitney U test as all the variables' distributions were skewed. Binary logistic regression was performed for each lipid fraction category to assess the association with PCOS and its manifestations, after adjustment for BMI and HOMA-IR status. Any p-value below 0.05 was considered statistically significant.

Result

The androgenic features (mFG score, acne, TT, FAI) were higher among women with PCOS than in the control group. Women with PCOS had poorer metabolic profiles (BP, acanthosis nigricans, glucose, lipid profile, and IR) than the control group (Table-I).

The risk categories of the four lipid fractions among women with PCOS are shown in Table-II. According to NCEP ATP III cut-offs, approximately 5.6%, 42.5%, 6.3%, 14.3%, and 23.3% of women with PCOS had high-risk TC, HDL, LDL, TG, and non-HDL cholesterol, respectively. Besides, nearly 21.3% had atherogenic dyslipidemia. Among all lipid fraction classifications, only the high-risk LDL-cholesterol category showed a significant association with PCOS after adjustment for BMI and HOMA-IR status.

Approximately 70.7% of women with PCOS had at least one abnormality in their lipid profile that was significantly higher than that of the control group. Women with PCOS had a higher frequency of all the components of dyslipidemia. However, a significant difference was found only with three components (Figure-1).

When we categorized the study population by BMI categories, only LDL cholesterol, TG, and non-HDL cholesterol significantly differed in lean participants; HDL cholesterol, TG, and non-HDL cholesterol in overweight participants; and none in obese participants

Table-I: Characteristics of the study participants (n= 477)

Variables	PCOS (n= 287)	Control (n= 190)	p
Age, years	24.0 (21.0 – 27.0)	24.0 (21.0 – 29.0)	0.743
Age of menarche, year	13.0 (12.0 – 13.0)	13.0 (12.0 – 13.0)	0.750
Body mass index, kg/m2	27.4 (24.4 – 30.8)	21.8 (19.5 – 25.3)	**<0.001**
Waist circumference, cm	88.0 (81.0 – 97.0)	76.0 (69.0 – 83.0)	**<0.001**
Systolic blood pressure, mm-Hg	115.0 (105.0 – 120.0)	110.0 (100.0 – 110.0)	**<0.001**
Diastolic blood pressure, mm-Hg	75.0 (70.0 – 80.0)	70.0 (60.0 – 76.3)	**<0.001**
Acne	157 (54.7)	10 (5.3)	**<0.001**
Acanthosis	186 (64.8)	6 (3.2)	**<0.001**
Modified Ferriman-Gallwey score	11.0 (8.0 – 15.0)	1.0 (0.0 – 2.0)	**<0.001**
Fasting plasma glucose, mmol/L	5.1 (4.8 – 5.5)	4.6 (3.9 – 5.1)	**<0.001**
Total cholesterol, mg/dL	179.0 (158.0 – 201.0)	165.0 (144.0 – 181.3)	**<0.001**
LDL-cholesterol, mg/dL	109.0 (94.8 – 130.0)	98.0 (83.0 – 114.3)	**<0.001**
HDL-cholesterol, mg/dL	41.0 (36.0 – 46.0)	45.0 (39.0 – 51.0)	**<0.001**
Non-HDL cholesterol, mg/dL	137.0 (117.0 – 159.0)	118.5 (101.0 – 137.3)	**<0.001**
Triglyceride, mg/dL	132.0 (100.0 – 173.0)	98.0 (83.0 – 114.3)	**<0.001**
Total testosterone, ng/dL	53.0 (33.9 – 81.5)	25.4 (19.0 – 35.2)	**<0.001**
Sex-hormone binding globulin, nmol/L	26.5 (19.7 – 34.5)	50.5 (43.2 – 62.7)	**<0.001**
Free androgen index, %	7.3 (4.1 – 12.5)	1.8 (1.3 – 2.5)	**<0.001**
Fasting insulin, µIU/mL	15.1 (10.5 – 23.6)	8.1 (5.9 – 10.8)	**<0.001**
Homeostatic model assessment-IR	3.5 (2.3 – 5.4)	1.6 (1.1 – 2.2)	**<0.001**

Mann-Whitney U test was done, Pearson's chi-square test was done

Table-II: Association of lipid fractions with PCOS (n= 477)

Lipid fractions	Risk category	PCOS, n= 287	Control, n= 190	p†	OR* (95% CI)	p*
Total cholesterol, mg/dL	Desirable, <200	210 (73.2)	166 (87.4)	**<0.001**	1	
	Borderline, 200 - 239	61 (21.3)	22 (11.6)		1.3 (0.7 – 2.5)	0.382
	High, ≥240	16 (5.6)	2 (1.1)		5.0 (1.0 – 25.5)	0.054
HDL cholesterol, mg/dL	Low risk, ≥60	13 (4.5)	11 (5.8)	**0.008**	1	
	Intermediate risk, 50 -59	152 (53.0)	125 (65.8)		0.7 (0.3 – 2.0)	0.528
	High risk, <40	122 (42.5)	54 (28.4)		1.1 (0.3 – 3.0)	0.914
LDL cholesterol, mg/dL	Desirable, <130	215 (74.9)	170 (89.5)	**<0.001**	1	
	Borderline, 130 - 159	54 (18.8)	19 (10.0)		1.6 (0.8 – 3.2)	0.152
	High, ≥160	18 (6.3)	1 (0.5)		9.2 (1.1 – 77.5)	**0.041**
Triglyceride, mg/dL	Desirable, <150	181 (63.1)	160 (84.2)	**<0.001**	1	
	Borderline, 150 - 200	65 (22.6)	22 (11.6)		1.1 (0.6 – 2.1)	0.698
	High, ≥200	41 (14.3)	8 (4.2)		1.1 (0.4 – 2.7)	0.866
Non-HDL cholesterol, mg/dL	Desirable, <160	220 (76.7)	172 (90.5)	**<0.001**	1	
	Undesirable, ≥160	67 (23.3)	18 (9.5)		1.8 (0.9 – 3.5)	0.087
Isolated low HDL cholesterol	Absent	226 (78.7)	153 (80.5)	0.637		
	Present	61 (21.3)	37 (19.5)		1.6 (0.9 – 2.8)	0.111
Atherogenic dyslipidemia	Absent	226 (78.7)	173 (91.1)	**<0.001**	1	
	Present	61 (21.3)	17 (8.9)		1.0 (0.5 – 2.1)	0.916

†Pearson's chi-squared test was done. *Binary logistic regression analysis with study groups as dependent variable after adjustment for BMI and HOMA-IR categories

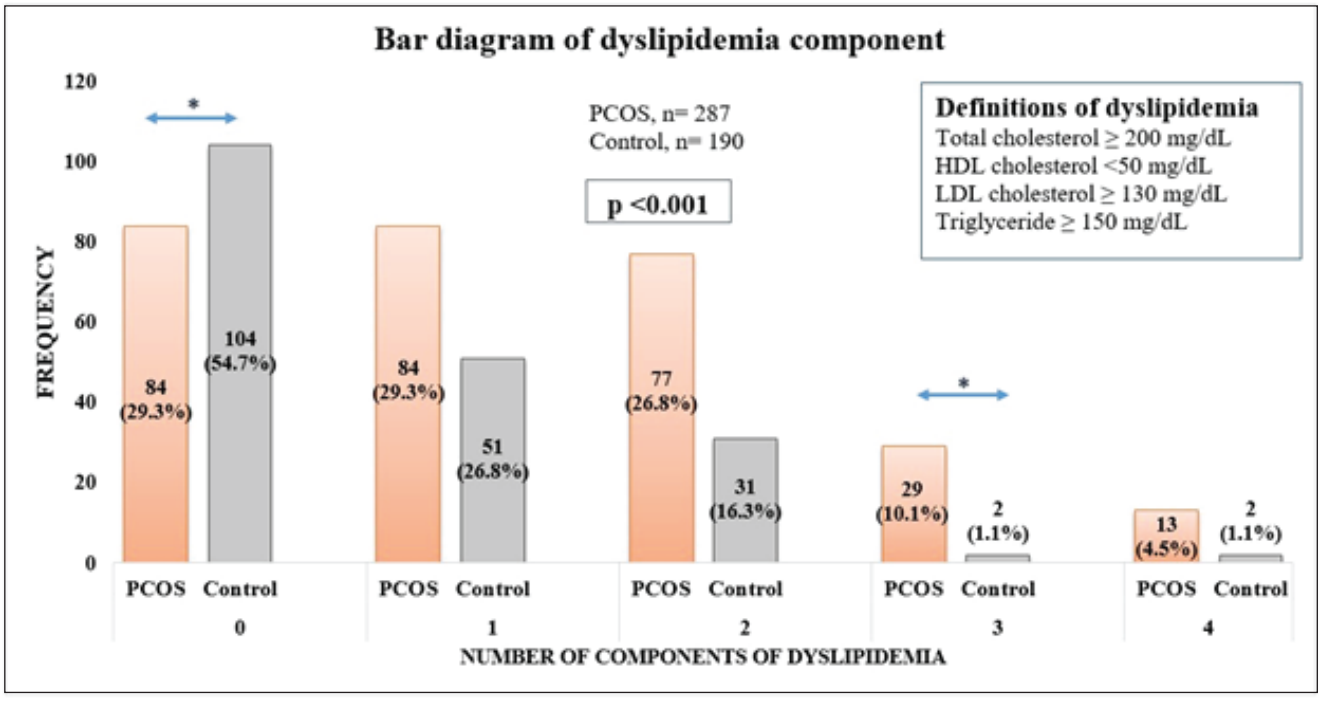

Figure-1: Number of dyslipidemia components between the study groups (n= 477)

Pearson's chi-squared test with post hoc from adjusted residuals was done

between the study groups (Figure-2). Similarly, by HOMA-IR categories, all the lipid fractions significantly varied only in those with insulin-sensitive subgroups but not in the insulin-resistant subgroup between the study groups (Figure-3).

None of the diagnostic features, including phenotypes,

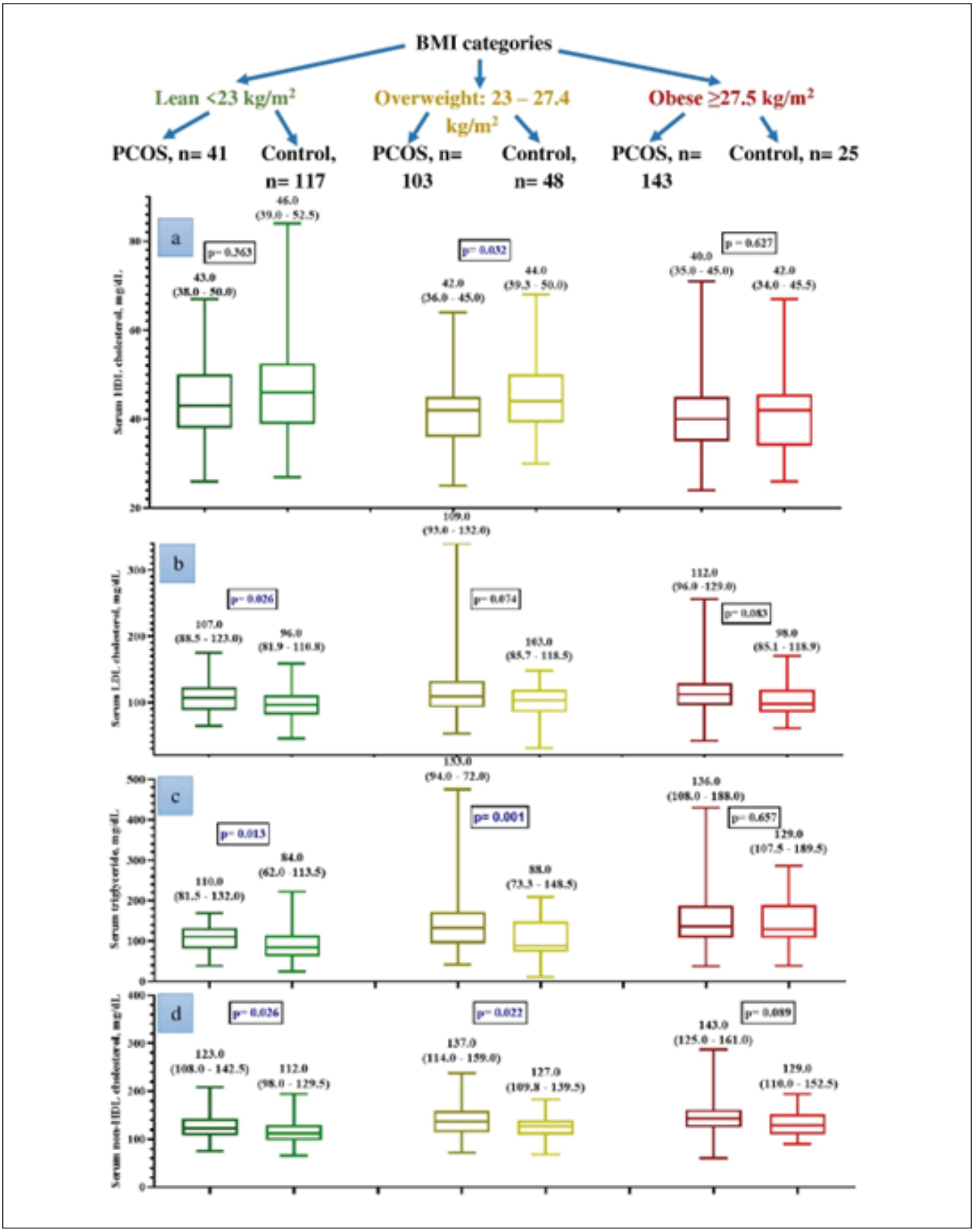

Figure-2. Lipid fractions according to BMI status between the study groups (n= 477)

Mann-Whitney U test was done

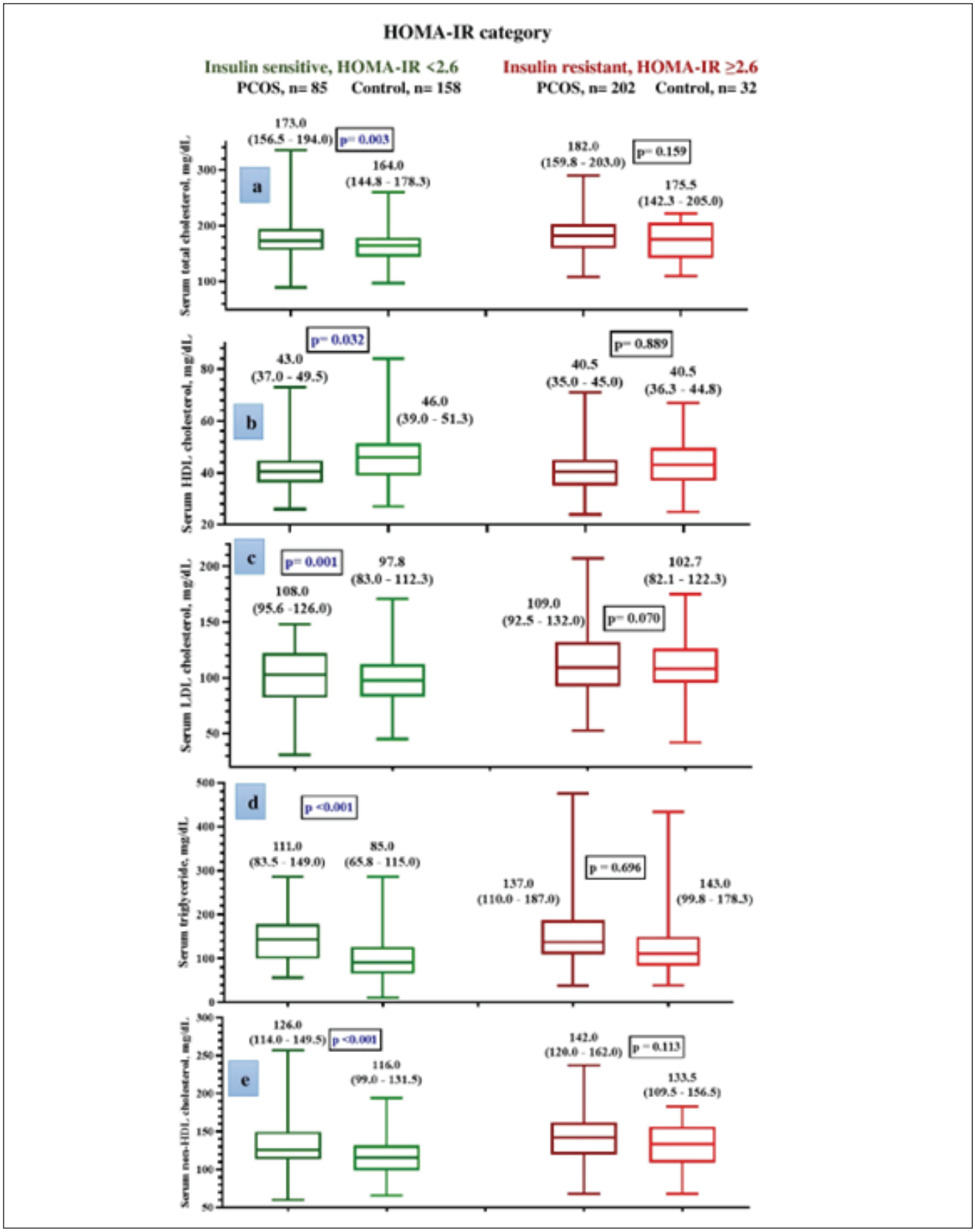

Figure-3. Lipid fractions according to HOMA-IR status between the study groups (n= 477)

Mann-Whitney U test was done

Table-III: Association of different characteristics of PCOS with undesirable category of lipid fractions (n= 287)

Variables	Total cholesterol ≥200 mg/dL (n: 77 vs. 210)		LDL-cholesterol ≥130 mg/dL (n: 72 vs. 215)		HDL-cholesterol <50 mg/dL (n: 122 vs. 165)		Triglyceride ≥150 mg/dL (n: 106 vs. 181)	
	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p	OR (95% CI)	p
Irregular cycle	1.4 (0.4 – 5.3)	0.582	1.3 (0.4 – 4.9)	0.666	3.1 (0.8 – 11.1)	0.089	1.3 (0.4 – 4.3)	0.707
Significant hirsutism	0.8 (0.4 – 1.7)	0.492	1.0 (0.4 – 2.2)	0.926	0.8 (0.4 – 1.5)	0.426	0.5 (0.2 – 1.1)	0.101
Hyperandrogenemia	0.9 (0.5 – 1.5)	0.552	0.8 (0.5 – 1.4)	0.423	1.4 (0.9 – 2.3)	0.183	1.6 (0.9 – 2.8)	0.085
PCOM	1.0 (0.5 – 2.0)	0.965	0.9 (0.5 – 1.8)	0.722	1.2 (0.7 – 2.3)	0.541	0.9 (0.5 – 1.7)	0.675
Phenotypes*								
A	0.6 (0.2 – 1.6)	0.291	1.0 (0.3 – 3.2)	0.951	0.8 (0.3 – 2.1)	0.589	0.7 (0.2 – 2.2)	0.539
B	0.6 (0.2 – 1.9)	0.376	1.1 (0.3 – 3.9)	0.912	0.6 (0.2 – 1.8)	0.367	0.8 (0.2 – 2.8)	0.744
C	0.4 (0.1 – 2.1)	0.283	0.7 (0.1 – 4.1)	0.731	0.2 (0.1 – 1.2)	0.080	0.6 (0.1 – 3.0)	0.512

Binary logistic regression analysis was done after adjustment for BMI and HOMA-IR statuses
*The reference category was phenotype D

had independent associations with lipid fraction categories after adjustment for BMI and HOMA-IR statuses (Table-III).

Discussion

Dyslipidemia is the most common metabolic abnormality among women with PCOS.[7] However, we found that only those with severe LDL cholesterol had a significant association with PCOS after adjustment for BMI and HOMA-IR statuses. Different lipid fractions varied between the study groups, only among those without obesity and insulin resistance. There were no significant associations between lipid-fraction categories and different features of PCOS.

Wild et al. (2011) found higher TC, LDL-cholesterol, and TG levels, but lower HDL cholesterol levels, among women with PCOS than among healthy controls in their meta-analysis.[12] We also found this set of classic abnormalities in lipid profiles among women with PCOS. Similarly, a meta-analysis of Brazilian studies also confirmed these findings.[19] However, another recent meta-analysis found only higher TC and lower HDL cholesterol among women with PCOS than controls.[20]

Around 71% of women with PCOS had at least one abnormality in their lipid profile in our study. Similarly, a study conducted in South India showed abnormalities in 70% of PCOS women.[21] A lower prevalence (41%) was reported in China.[22] Low HDL cholesterol was the most common dyslipidemia (42.5%) among women with PCOS in our study. Similar findings but at different rates were reported by many studies.[21-24]

Obesity, IR, and androgen status are essential determinants of lipid profile in PCOS. In our study, lipid fractions did not differ significantly with androgen status or phenotypes. We included only BMI and HOMA-IR status in the regression model to examine the independent association between PCOS and lipid fractions. Among them, only the high-risk LDL cholesterol category showed a predictive association with PCOS. It is thought that higher LDL cholesterol levels are a unique abnormality among women with PCOS, driven by hyperandrogenism rather than IR.[25] A previous study found a significant association between LDL and non-HDL cholesterol in PCOS after matching only on BMI.[12] In their meta-analysis, Zhuang et al. (2022) divided the study population (18 – 45 years) into three groups based on the BMI cut-off of 25 and 30 kg/m^2. Serum TG, LDL-cholesterol, and non-HDL-cholesterol increased in all BMI groups, and HDL-cholesterol decreased only in those with a BMI below 30 kg/m^2 in women with PCOS than in the matched controls.[26] Similarly, we did not find significant differences between the women with obesity and insulin resistance groups. We showed previously higher total and LDL-cholesterol levels and hypertriglyceridemia even in women with lean PCOS than matched controls.[27] Hence, lipid profiles should be checked even in non-obese, insulin-sensitive women with PCOS. Moreover, they should be followed up periodically for early detection of atherosclerotic cardiovascular diseases. It may be that, at lower BMI and insulin-sensitive states, lipid abnormalities depend more on androgens, and that they become more dependent on increased BMI and HOMA-IR levels and the association loss.[28] The frequency of low HDL cholesterol was significantly higher among hyperandrogenic phenotypes than phenotype D in a meta-analysis.[29] However, we did not find this association. This discrepancy might be

explained by the heterogeneous nature of PCOS in different races.

The main limitation of our study was its conduct in a single tertiary care center. Convenience sampling and small sample sizes during subgroup analyses further reduce its applicability in generalization. Besides, we could not measure lipoproteins or small, dense LDL particles, which might be more closely related to CV risk.[25] Moreover, the number of control women was lower than that of PCOS.

Conclusions

Dyslipidemia is a common metabolic abnormality among women with PCOS that depends on BMI and IR, except for LDL cholesterol, with an independent association. When the BMI and IR exceed a certain threshold, the association between lipid fractions and PCOS is lost. However, the lipid fractions did not affect the diagnostic features and phenotypes of PCOS. Early detection, prompt management, and regular follow-up for these abnormalities may be helpful for future prevention of CV events among women with PCOS.

Conflict of interest

The authors have no conflicts of interest to disclose.

Acknowledgements

We are grateful to the study participants and their attendants who actively cooperated during sample collection.

Disclosure

This study is not funded by any companies, NGOs, or institutions.

Financial Disclosure

This study did not receive any funding.

Data Availability

Any queries regarding this study should be directed to the corresponding author, and supporting data are available from the corresponding author upon reasonable request.

Ethical Approval and Consent to Participate

All procedures performed in this study involving human participants were conducted in accordance with the ethical standards and the 1964 Helsinki Declaration. Informed written consent was obtained from each of the participants included in the study. Ethical approval (R/N no: 3582; BSMMU/2021/7642, date: 24/08/2021) was obtained from the Institutional Review Board (IRB) of Bangladesh Medical University.

How to cite this article: Shahed-Morshed M, Banu H, Hasanat MA. Dyslipidemia varies among the metabolic phenotypes of polycystic ovary syndrome. J Assoc Clin Endocrinol Diabetol Bangladesh 2026; 5(1): 12-20

Publication History

Received on: 05 Oct 2025

Accepted on: 10 Dec 2025

Published on: 01 Jan 2026

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