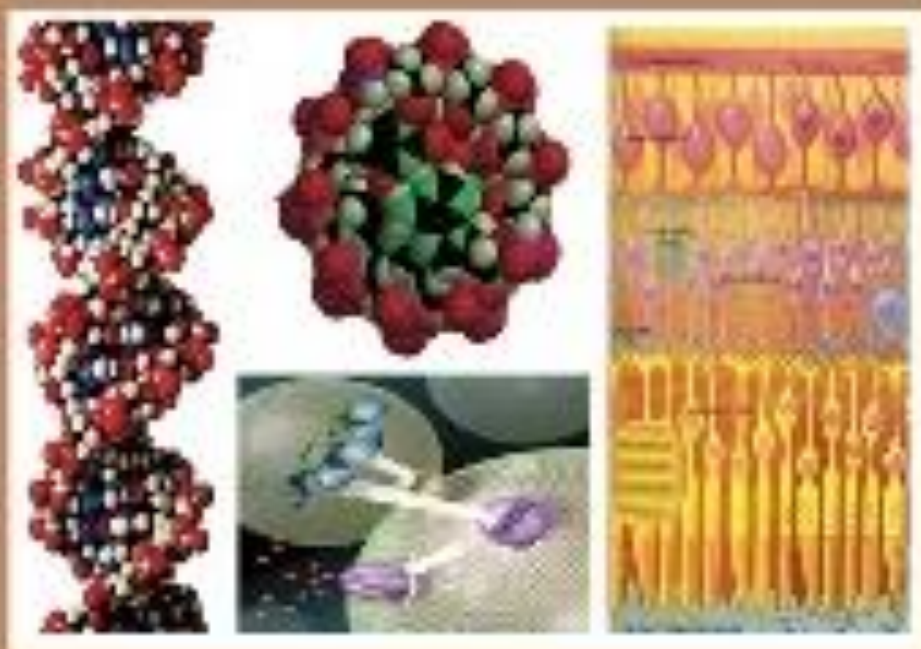




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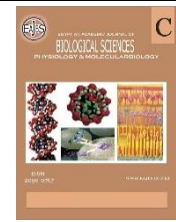
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Association of Body Mass Index with Metabolic and Physiological Parameters of Women with Polycystic Ovary Syndrome: A Cross-Sectional Study

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ABSTRACT

Background: Polycystic ovary syndrome is a common endocrine disorder associated with metabolic disturbances, including abnormal lipid levels and obesity. Clarifying the impact of weight status on metabolic factors in affected women is essential for effective management strategies. **Objectives:** This study aimed to evaluate the impact of body mass index on blood lipid levels, fasting blood sugar, and blood pressure in women with polycystic ovary syndrome, and to determine the correlations between these metabolic parameters and weight status. **Methods:** The study included women diagnosed with polycystic ovary syndrome according to the Rotterdam criteria. Data collection included demographic and clinical characteristics, as well as anthropometric (body mass index and blood pressure) and biochemical (fasting blood sugar and lipid profile) measurements. Body mass index was used to classify participants into normal weight, overweight, and obese categories based on the World Health Organization classification. Statistical analysis involved the Kruskal-Wallis test, the Dunn-Bonferroni post-hoc test, and Spearman's rank-order correlation to assess associations between body mass index and metabolic markers. **Results:** Among the participants, 69% were classified as either overweight or obese. A higher body mass index was significantly associated with adverse changes in lipid profile, including elevated triglyceride levels ($p = 0.00$) and low-density lipoprotein cholesterol ($p = 0.00$), along with reduced high-density lipoprotein cholesterol levels ($p = 0.00$). Systolic blood pressure was also significantly higher in overweight and obese individuals ($p = 0.04$). However, fasting blood sugar levels did not differ significantly across body mass index categories ($p = 0.19$). Correlation analysis revealed a positive association between body mass index and triglycerides ($\rho = 0.26$, $p = 0.00$) and low-density lipoprotein cholesterol ($\rho = 0.20$, $p = 0.04$), while body mass index was negatively correlated with high-density lipoprotein cholesterol ($\rho = -0.27$, $p = 0.00$). **Conclusion:** Body mass index is strongly associated with lipid abnormalities and elevated blood pressure in women with polycystic ovary syndrome, reinforcing the importance of weight management in reducing metabolic and cardiovascular risks.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a complex endocrine disorder affecting women of reproductive age, characterized by a constellation of hormonal, reproductive, and metabolic disturbances. It commonly presents with clinical features such as hyperandrogenism, including hirsutism, acne, and scalp hair thinning, alongside menstrual irregularities like oligomenorrhea or amenorrhea. Ultrasound may reveal polycystic ovarian morphology, marked by multiple small follicles (Azziz *et al.*, 2016).

Although not all individuals exhibit every feature, many are at increased risk of insulin resistance, type 2 diabetes, and cardiovascular disease (Su *et al.*, 2025). In addition to its physical effects, PCOS is associated with significant mental health concerns, including anxiety, depression, and a reduced quality of life (Allen *et al.*, 2022). Its global prevalence is estimated at 15–20%, underscoring its significance as a major women's health concern (Barnard *et al.*, 2007).

Dyslipidemia is one of the most prevalent metabolic disturbances observed in up to 70% of women with PCOS, significantly increasing the risk of developing coronary artery disease over time (Legro *et al.*, 2001; Wild *et al.*, 2011). Moreover, women with PCOS exhibit a heightened risk of atherosclerosis and cardiovascular complications, with studies indicating up to a seven-fold increase in the likelihood of myocardial infarction compared to the general population (Kumariya *et al.*, 2021). Cardiometabolic risks in PCOS are partly driven by elevated androgen levels and common obesity, both promoting metabolic disturbances. High androgens reduce lipoprotein lipase (LPL) activity in abdominal fat, leading to increased central adiposity (Talbot *et al.*, 2000).

Adiposity, particularly android fat distribution, is closely associated with the severity of symptoms in women with PCOS and related metabolic disturbances. Women with PCOS commonly exhibit higher body mass index (BMI) and increased waist circumference (WC), which are important, cost-effective anthropometric measures widely used to evaluate the risk of non-communicable diseases such as diabetes, hypertension, and dyslipidemia (Keyif and Yavuzcan, 2025). Obesity has been identified as a significant determinant of cardiovascular and metabolic complications in PCOS (Shen *et al.*, 2015), a condition that shares multiple risk factors with cardiovascular diseases. While limited studies have focused on the correlation between lipid profiles and

anthropometric measures in PCOS, understanding these associations is critical. This study investigates the relationship between anthropometric measurements and metabolic parameters, including lipid profile, fasting blood sugar (FBS), and blood pressure (BP), in women with PCOS in Duhok City, Kurdistan Region of Iraq. Examining whether these anthropometric parameters are associated with metabolic factors and highlighting the importance of early intervention and personalized treatment strategies for managing these interconnected conditions. By deepening our understanding of these relationships, this research seeks to improve clinical outcomes and enhance the quality of life for women affected by this complex syndrome.

MATERIALS AND METHODS

Study Design and Participant Selection:

A cross-sectional study was carried out at the Gynaecology Department of Azadi Teaching Hospital in Duhok, Kurdistan Region, Iraq, between October 2024 and February 2025. A total of 100 women, aged between 14 and 40 years, were recruited through consecutive non-probability sampling. Ethical approval for the study was obtained before data collection from the General Directorate of Health in Duhok (Ref: 25092024-8-17), and all participants provided written informed consent before their inclusion. The enrolled women met the diagnostic criteria for PCOS based on the 2003 Rotterdam guidelines established by the European Society for Human Reproduction and Embryology (ESHRE) and the American Society for Reproductive Medicine (ASRM) (Fauser, 2004). Participants included both newly diagnosed cases identified during the study and women with a prior clinical diagnosis of PCOS, provided they fulfilled the specified diagnostic criteria at the time of enrollment. Diagnosis required the presence of at least two of the following criteria: (1) Oligo- or anovulation, defined as menstrual cycles exceeding 35 days or the absence of menstruation for three months or more; (2) clinical or biochemical evidence of

hyperandrogenism, such as excessive hair growth, acne, or elevated serum testosterone levels; and (3) polycystic ovarian morphology on ultrasound, characterized by more than 10 subcapsular follicles measuring 2–8 mm in diameter, accompanied by increased stromal density. Women with diabetes mellitus, Cushing's syndrome, congenital adrenal hyperplasia, autoimmune diseases, or chronic inflammatory conditions were excluded from the study. Additionally, women with a known history of hypertension or those who had used hormonal therapies, ovulation-inducing agents, anti-androgen medications, or lipid-lowering drugs within the past three months were excluded. Participants currently using any medications known to significantly affect metabolic or hormonal parameters, such as corticosteroids, antipsychotics, or other drugs impacting lipid profile, glucose metabolism, or endocrine function, were also excluded to minimize confounding effects.

Clinical and Biochemical Data Collection:

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured once using a validated automated electronic sphygmomanometer after the participant had rested in a seated position for 10 minutes, following a standardized protocol appropriate for cross-sectional studies. Anthropometric measurements, including height and weight, were recorded to calculate BMI (kg/m^2), which was categorized based on the World Health Organization (WHO) (2000) classification: Normal weight ($18.5\text{--}24.9 \text{ kg}/\text{m}^2$), overweight ($25\text{--}29.9 \text{ kg}/\text{m}^2$), and obesity ($\geq 30 \text{ kg}/\text{m}^2$). Following a 12-hour overnight fast, 4 mL of venous blood was collected from the cubital vein using standard phlebotomy procedures. The blood samples were drawn into plain gel tubes, allowed to clot at room temperature for 30 minutes, and then centrifuged at 3000 rpm for 10 minutes at 4°C to obtain serum. Biochemical analyses, including FBS and lipid profile parameters, total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), were performed using the Cobas c 501 module (Roche 6000 Diagnostics,

Germany). Lipid levels were interpreted according to local reference ranges provided by the Central Health Laboratory in Duhok, where $\text{TC} > 200 \text{ mg}/\text{dL}$, $\text{TG} > 150 \text{ mg}/\text{dL}$, and $\text{LDL-C} > 100 \text{ mg}/\text{dL}$ were considered elevated, while HDL-C was classified as low if below $40 \text{ mg}/\text{dL}$.

Statistical Analysis:

The normality of continuous variables was assessed using the Shapiro-Wilk test. Descriptive statistics were presented as frequencies and percentages. Differences between groups were compared using the Kruskal-Wallis H test for non-parametric variables, followed by post-hoc analysis with the Dunn-Bonferroni test. A p-value of less than 0.05 was considered statistically significant. Spearman's rank correlation coefficient was used for correlation analysis. The data were analysed using SPSS (version 25).

RESULTS

Demographic and clinical information, including age, marital status, family history of PCOS, infertility (assessed among married participants), menstrual irregularities, acne, and hirsutism, was collected from all participants. The key findings are summarized in Table 1. A majority (62.0%) resided in urban areas, with 47.0% unemployed and 36.0% students. Most participants (75.0%) were unmarried, while 25.0% were married, among whom 76.0% reported subfertility. A family history of PCOS was noted in 42.0% of cases. Menstrual irregularities were prevalent, with oligomenorrhea in 48.0% of participants, amenorrhea in 39.0%, and regular cycles in only 13.0%. Hyperandrogenic features were common, with hirsutism affecting 89.0% and acne observed in 69.0%. Regarding body weight, 38.0% of participants were overweight, 31.0% were obese, and 31.0% had a normal weight. The median age was 23 years (IQR: 20–30 years). Additional detailed anthropometric, clinical, and biochemical parameters are comprehensively presented in Table 2, offering further insight into the metabolic and physiological characteristics of the study population.

Table 1. Demographic and Clinical Characteristics of Participants

Variable	Category	Frequency	Percent (%)
Residence	Rural	38	38.0
	Urban	62	62.0
Occupation	Employed	17	17.0
	Student	36	36.0
	Unemployed	47	47.0
Marital State	Married	25	25.0
	Unmarried	75	75.0
Family History of PCOS	No	58	58.0
	Yes	42	42.0
Cycle Pattern	Amenorrhea	39	39.0
	Oligomenorrhea	48	48.0
	Regular	13	13.0
Subfertility	No	6	24.0
	Yes	19	76.0
Hirsutism	No	11	11.0
	Yes	89	89.0
Acne	No	31	31.0
	Yes	69	69.0
BMI (kg/m ²)	Normal Weight	31	31.0
	Overweight	38	38.0
	Obese	31	31.0

Table 2: Descriptive statistics of anthropometric, clinical, and biochemical parameters.

Variable	Mean \pm SD	Median (IQR)	Minimum – Maximum
Age (Years)	24.87 \pm 6.14	23.00 (20.00–30.00)	14 – 37
SBP (mmHg)	115.16 \pm 16.70	113.50 (103.00–123.00)	83 – 168
DBP (mmHg)	73.36 \pm 12.77	72.50 (65.25–78.00)	40 – 129
Height (m)	1.59 \pm 0.07	1.60 (1.53–1.64)	1.42 – 1.75
Weight (kg)	70.00 \pm 13.40	69.25 (60.00–79.25)	45.0 – 75.0
BMI (kg/m ²)	27.72 \pm 5.22	27.08 (23.78–31.48)	18.14 – 40.23
FBS (mg/dL)	92.30 \pm 9.81	92.00 (86.00–99.00)	66 – 132
TC (mg/dL)	166.19 \pm 32.99	165.00 (141.00–189.75)	78 – 259
TG (mg/dL)	105.69 \pm 42.55	95.50 (77.00–133.25)	37 – 220
LDL (mg/dL)	101.59 \pm 29.89	100.00 (81.00–117.00)	43 – 178
HDL (mg/dL)	47.92 \pm 10.75	44.15 (41.00–54.75)	27.0 – 75.0

SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, BMI: Body Mass Index, FBS: Fasting Blood Sugar, TC: Total Cholesterol, TG: Triglycerides, LDL: Low-Density Lipoprotein, HDL: High-Density Lipoprotein, SD: standard deviation, IQR: interquartile range.

A significant prevalence of lipid abnormalities was observed among the participants. Elevated LDL-C levels were found in 47.0% of the participants, while 18.0% exhibited low HDL-C levels. Additionally, elevated TG and TC were

present in 15.0% and 16.0% of participants, respectively. These findings underscore the considerable proportion of individuals with dyslipidaemia, especially those with elevated LDL-C levels (Fig. 1).

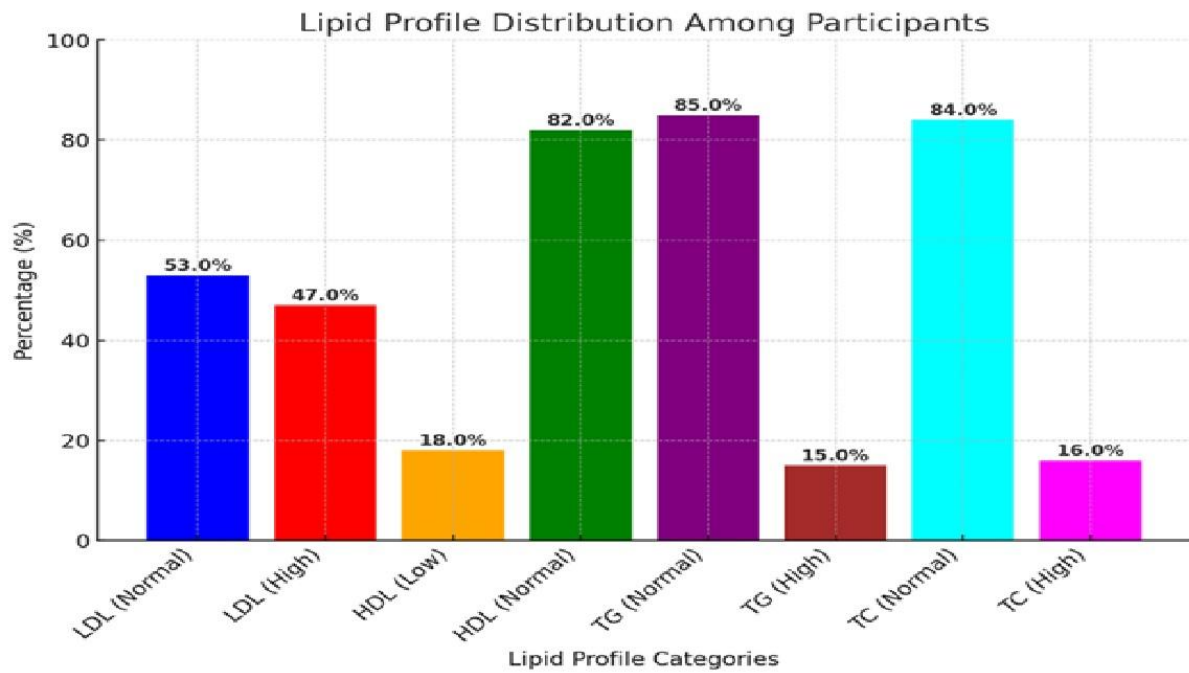


Fig. 1: Prevalence of Lipid Abnormalities Among Study Participants.

This figure illustrates the proportion of participants exhibiting abnormal lipid levels according to clinical cut-off values. The assessed parameters include TG (triglycerides), LDL-C (low-density lipoprotein cholesterol), HDL-C (high-density lipoprotein cholesterol), and TC (total cholesterol).

The data from this study reveal significant differences in several metabolic and cardiovascular parameters across weight categories (Table 3). SBP increased significantly with weight gain ($p = 0.04$). Median SBP values rose from 106 mmHg (IQR: 98–119) in normal-weight individuals to 117 mmHg in both the overweight (IQR: 106–123) and obese (IQR: 107–127) groups. Alterations in the lipid profile were also significantly associated with weight status. TG levels increased significantly with weight gain ($p = 0.00$), with higher median values observed in overweight (103 mg/dL, IQR: 76.5–147.75) and obese individuals (108 mg/dL, IQR: 82–144) compared to the normal-weight group (85 mg/dL, IQR: 59–105). Similarly, LDL-C levels were significantly higher in overweight (101

mg/dL, IQR: 82–123.25) and obese individuals (104 mg/dL, IQR: 95–127) compared to normal-weight individuals (86 mg/dL, IQR: 66–112, $p = 0.00$). In contrast, HDL-C levels showed a significant decline ($p = 0.000$), decreasing from 53 mg/dL (IQR: 41–65) in normal-weight individuals to 45 mg/dL (IQR: 40.98–54.25) in overweight and 43 mg/dL (IQR: 40–48) in obese individuals. Although FBS and TC exhibit a trend toward worsening with weight gain, their differences were not statistically significant ($p = 0.19$ and $p = 0.28$, respectively). DBP also does not differ significantly across groups ($p = 0.33$), with median values of 68 mmHg (IQR: 64–77) in normal-weight individuals, 75.5 mmHg (IQR: 67–80.5) in overweight individuals, and 74 mmHg (IQR: 65–82) in obese individuals.

Table 3: Comparison of Clinical and Metabolic Parameters Across BMI Categories:

Variable	Normal Weight	Overweight	Obese	p-value
	Median, IQR			
SBP (mmHg)	106 (98–119)	117 (106–123)	117 (107–127)	0.04*
DBP (mmHg)	68 (64–77)	75.5 (67–80.5)	74 (65–82)	0.33
FBS (mg/dL)	90 (83–95)	92.5 (86–100)	91 (85–100)	0.19
TC (mg/dL)	147 (137–177)	168 (155–192)	168 (152.5–190)	0.28
TG (mg/dL)	85 (59–105)	103 (76.5–147.75)	108 (82–144)	0.00*
HDL-C (mg/dL)	53 (41–65)	45 (40.98–54.25)	43 (40–48)	0.00*
LDL-C (mg/dL)	86 (66–112)	101 (82–123.25)	104 (95–127)	0.00*

SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, FBS: Fasting Blood Sugar, TC: Total Cholesterol, TG: Triglycerides, LDL: Low-Density Lipoprotein, HDL: High-Density Lipoprotein. Two-tailed $p < 0.05$ is considered significant. *Post hoc analysis using Dunn-Bonferroni correction revealed the following:

- **TG:** Normal-weight individuals have significantly different TG levels compared to both the overweight ($p = 0.016$) and obese ($p = 0.006$) groups after adjustment.
- **HDL:** A statistically significant difference was found between the obese and normal-weight groups ($p = 0.021$ after adjustment).
- **LDL:** The normal-weight group significantly differs from the obese group ($p = 0.025$ after adjustment).
- **SBP:** No significant difference was found between the normal-weight and overweight groups or between the overweight and obese groups after Bonferroni correction.

Table 4, illustrates a notable positive association between BMI and TG ($\rho = 0.26$, $p = 0.00$) as well as LDL-C ($\rho = 0.20$, $p = 0.04$). Conversely, BMI was negatively correlated with HDL-C ($\rho = -0.27$, $p = 0.00$). A strong positive correlation was also observed between body weight and TG ($\rho = 0.29$, $p = 0.00$). However, no significant

correlations were found between weight and TC, HDL-C, or LDL-C. Additionally, age was positively correlated with LDL-C levels ($\rho = 0.24$, $p = 0.01$), suggesting an increase in LDL-C levels with age. A negative correlation was also noted between age and HDL-C ($\rho = -0.20$, $p = 0.04$).

Table 4: Correlation Between Anthropometric Measures and Metabolic Parameters in Women With PCOS.

Variable		TC	TG	HDL-C	LDL-C	SBP	DBP	FBS
Age (years)	ρ	0.16	0.16	-0.20*	0.24*	0.10	0.09	-0.07
	p	0.10	0.10	0.04	0.01	0.28	0.36	0.48
Weight (kg)	ρ	0.13	0.29**	-0.17	0.15	0.04	0.15	0.12
	p	0.19	0.00	0.09	0.13	0.67	0.12	0.22
Height (m)	ρ	0.08	0.10	0.19	-0.08	0.23*	0.07	0.07
	p	0.38	0.32	0.05	0.41	0.01	0.45	0.45
BMI (kg/m ²)	ρ	0.09	0.26**	-0.27**	0.20*	0.23*	0.13	0.08
	p	0.32	0.00	0.00	0.04	0.01	0.17	0.38

ρ = Spearman's correlation coefficient; p -values are two-tailed; * = $p < 0.05$ (statistically significant); ** = $p < 0.01$ (highly statistically significant); TC = Total Cholesterol; TG = Triglycerides; HDL-C = High-Density Lipoprotein Cholesterol; LDL-C = Low-Density Lipoprotein Cholesterol; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; FBS = Fasting Blood Sugar; BMI = Body Mass Index.

DISCUSSION

This study provides a comprehensive evaluation of the metabolic, clinical, and demographic characteristics of reproductive-aged women diagnosed with PCOS. It also identifies important clinical and

demographic patterns in PCOS patients. The age distribution revealed that most participants were between 20 and 30 years old, which is consistent with the age range commonly associated with the clinical manifestation and diagnosis of PCOS, as

reported by (Akshaya and Bhattacharya, 2016). During this period, symptoms typically become more evident, increasing the likelihood of medical consultation, particularly due to infertility-related issues.

The relationship between PCOS and obesity is bidirectional, with each condition exacerbating the other in a continuous cycle that perpetuates metabolic and hormonal disturbances (Ehrmann, 2005). Studies show that between 30% and 75% of women with PCOS are categorized as obese (Essah and Nestler, 2006; Glueck *et al.*, 2005; Rojas *et al.*, 2014). In this study, a significant proportion of participants had a BMI not within the normal range, with 69% of women with PCOS being either overweight or obese. Specifically, 31% of the women were classified as obese, while nearly 38% were overweight. Among the commonly used parameters for assessing obesity, BMI and waist circumference are the most recognized, with the latter providing a more accurate measure of visceral adiposity (Borrueal *et al.*, 2014), but BMI was found to be a more reliable indicator for identifying obesity in women with PCOS (Rasool, 2021). Accordingly, BMI was used in this study as the primary measure for assessing obesity-related metabolic disturbances in PCOS.

The study highlights a strong association between BMI and lipid profile abnormalities in women with PCOS, emphasizing BMI role in metabolic disturbances. Specifically, BMI was positively correlated with TG and LDL-C levels, while it showed a negative correlation with HDL-C. These findings align with a meta-analysis conducted by Wild *et al.*, which identified a significant relationship between BMI, LDL-C, and non-HDL-C in women with PCOS (Wild *et al.*, 2011). Similarly, Rocha *et al.* demonstrated that BMI had a notable impact on HDL-C levels in this population (Rocha *et al.*, 2011). While our study did not establish a significant correlation between BMI and TC, Saghafi-Asl *et al.* reported a positive association between BMI and both TC and LDL-C (Saghafi-asl *et al.*, 2013). Notably,

overweight and obese women exhibited elevated TG and LDL-C levels, indicating an increased susceptibility to dyslipidaemia, one of the key contributors to metabolic syndrome and cardiovascular disease. Additionally, a decline in HDL-C, a protective lipoprotein, further exacerbates cardiovascular risk, as lower HDL-C levels are associated with impaired cholesterol clearance and heightened atherogenesis. A study on Indian PCOS patients by Kar *et al.* and Sujatha *et al.* had similarly reported a high prevalence of obesity and lipid abnormalities in women with PCOS, reinforcing the role of obesity in disrupting lipid metabolism (Kar, 2013; Thathapudi *et al.*, 2014).

The findings of this study align with those of Manjunatha *et al.*, who reported significant increases in serum TG, TC, LDL-C, and very low-density lipoprotein (VLDL-C) levels, along with a decline in HDL-C in women with PCOS (Bennal *et al.*, 2014). Dyslipidaemia has been increasingly recognized as a common metabolic disorder in young adult women with PCOS (Vine *et al.*, 2023). The underlying mechanisms of dyslipidaemia in PCOS are multifaceted, with insulin resistance playing a central role. Insulin resistance promotes dyslipidaemia by enhancing lipolysis, altering lipoprotein lipase and hepatic lipase activity, and increasing the catabolism of HDL-C while promoting LDL-C formation. In addition to insulin resistance, hyperandrogenism contributes to lipid abnormalities by stimulating hepatic lipase activity, leading to excessive HDL-C catabolism.

Hyperinsulinemia and hyperandrogenemia further exacerbate metabolic dysfunction by promoting increased lipolysis in adipocytes, resulting in elevated free fatty acid release into circulation. This triggers an increase in hepatic VLDL-C secretion, contributing to hypertriglyceridemia. Consequently, the reverse cholesterol transport pathway is disrupted, leading to reduced HDL-C and elevated LDL-C levels (Cinar *et al.*, 2011). The rise in TG may also be attributed to increased lipogenesis, reduced clearance, or impaired fatty acid oxidation, all

of which further contribute to adiposity in women with PCOS.

Ethnic and geographical factors play a significant role in the variation of dyslipidaemia among women with PCOS. A study indicated that American women with PCOS have higher BMI and TG levels than Italian women (Essah *et al.*, 2008), while Korean women with PCOS experience a notable prevalence of dyslipidaemia, even in the absence of obesity (Kim and Choi, 2013). This suggests that lipid abnormalities are influenced by more than just body weight, with genetic predisposition, dietary patterns, and physical activity levels being key contributors. Recent findings by (Kim and Lee, 2022) demonstrate that lifestyle interventions, such as calorie restriction and regular physical activity, can significantly improve lipid profiles and overall metabolic health in women with PCOS.

Our study found no significant difference in FBS levels between normal-weight and overweight/obese women with PCOS. This contrasts with the findings of Mirdamadi *et al.*, who reported significantly higher FBS levels in obese women with PCOS compared to their lean counterparts (Mirdamadi *et al.*, 2020).

Several studies have reported an increased prevalence of hypertension in women with PCOS (Wu *et al.*, 2020; Zhuang *et al.*, 2022). Supporting this, our study found significantly higher SBP in obese women with PCOS. The heightened risk of hypertension in PCOS is thought to stem from a combination of factors such as hyperandrogenism, insulin resistance, obesity, and autonomic dysfunction. Specifically, the hyperandrogenic condition typical of PCOS contributes to cardiovascular risk by causing endothelial dysfunction, which may raise blood pressure levels (Marbaniang, 2023). Nevertheless, findings are not entirely consistent; (Mirdamadi *et al.*, 2020) observed that some obese women with PCOS maintain normal blood pressure, indicating that hypertension risk can vary among individuals in this population. This study has some limitations, including a small

sample size and the absence of a control group, which may limit the generalizability of the findings. Additionally, differences in PCOS phenotypes were not explored, which might have had some influence on the lipid and metabolic profiles. Future research should include larger samples, control groups, and consideration of PCOS phenotypes to better clarify metabolic variations.

Conclusion

This study demonstrates that increasing body weight in women with PCOS is significantly associated with adverse changes in lipid profiles and systolic blood pressure. Overweight and obese individuals exhibited higher TG and LDL-C levels, lower HDL-C levels, and elevated SBP compared to their normal-weight counterparts. While FBS, TC, and DBP did not differ significantly across weight groups, the overall metabolic pattern suggests a heightened cardiovascular risk in individuals with higher BMI. Correlation analysis further supports these findings, showing that BMI was positively associated with TG and LDL-C, and negatively associated with HDL-C. Similarly, body weight was positively correlated with TG levels. These results underscore the metabolic impact of obesity in women with PCOS and highlight the importance of weight management and cardiovascular risk monitoring in this population.

Declarations:

Ethical Approval: This study was approved by the General Directorate of Health in Duhok, Kurdistan Region, Iraq (Approval No: 25092024-8-17).

Competing interests: The authors have no competing interests to declare that are relevant to the content of this article.

Contributions: I hereby verify that all authors mentioned on the title page have made substantial contributions to the conception and design of the study, have thoroughly reviewed the manuscript, confirm the accuracy and authenticity of the data and its interpretation, and consent to its submission

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Availability of Data and Materials: All

datasets analysed and described during the present study are available from the corresponding author upon reasonable request.

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REFERENCES

- Allen, L.A., Shrikrishnapalasuriyar, N. and Rees, D.A. (2022), “Long-term health outcomes in young women with polycystic ovary syndrome: A narrative review”, *Clinical Endocrinology*, John Wiley and Sons Inc, Vol. 97 No. 2, pp. 187–198, doi: 10.1111/CEN.14609.
- Azziz, R., Carmina, E., Chen, Z., Dunaif, A., Laven, J.S.E., Legro, R.S., Lizneva, D., *et al.* (2016), “Polycystic ovary syndrome”, *Nature Reviews Disease Primers*, Nature Publishing Group, Vol. 2, doi: 10.1038/NRDP.2016.57.
- Barnard, L., Ferriday, D., Guenther, N., Strauss, B., Balen, A.H. and Dye, L. (2007), “Quality of life and psychological well being in polycystic ovary syndrome”, *Human Reproduction*, Vol. 22 No. 8, pp. 2279–2286, doi: 10.1093/HUMREP/DEM108.
- Bennal, A.S., Hiremath, S. and C, V.H. (2014), “ISSN 2347-954X (Print) Effect of PCOS on Lipid Profile”, *Scholars Journal of Applied Medical Sciences (SJAMS)*, Vol. 2 No. 3D, pp. 1153–1155, doi: 10.36347/sjams.2014.v02i03.058.
- Borrueal, S., Moltó, J.F., Alpañés, M., Fernández-Durán, E., Álvarez-Blasco, F., Luque-Ramírez, M. and Escobar-Morreale, H.F. (2014), “Surrogate markers of visceral adiposity in young adults: waist circumference and body mass index are more accurate than waist hip ratio, model of adipose distribution and visceral adiposity index”, *PloS One*, Vol. 9 No. 12, doi: 10.1371/JOURNAL.PONE.0114112.
- Cinar, N., Kizilarlanoglu, M.C., Harmanci, A., Aksoy, D.Y., Bozdog, G., Demir, B. and Yildiz, B.O. (2011), “Depression, anxiety and cardiometabolic risk in polycystic ovary syndrome”, *Human Reproduction (Oxford, England)*, Vol. 26 No. 12, pp. 3339–3345, doi: 10.1093/HUMREP/DER338.
- Ehrmann, D.A. (2005), “Polycystic Ovary Syndrome”, *New England Journal of Medicine*, Vol. 352 No. 12, pp. 1223–1236, doi: 10.1056/NEJMRA041536.
- Essah, P.A. and Nestler, J.E. (2006), “The metabolic syndrome in polycystic ovary syndrome”, *Journal of Endocrinological Investigation*, Vol. 29 No. 3, pp. 270–280, doi: 10.1007/BF03345554/METRICS.
- Essah, P.A., Nestler, J.E. and Carmina, E. (2008), “Differences in dyslipidemia between American and Italian women with polycystic ovary syndrome”, *Journal of Endocrinological Investigation*, Vol. 31 No. 1, pp. 35–41, doi: 10.1007/BF03345564.
- Fausser, B.C.J.M. (2004), “Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome”, *Fertility and Sterility*, Vol. 81 No. 1, pp. 19–25, doi: 10.1016/j.fertnstert.2003.10.004.
- Glueck, C.J., Dharashivkar, S., Wang, P., Zhu, B., Gartside, P.S., Tracy, T. and Sieve, L. (2005), “Obesity and extreme obesity, manifest by ages 20–24 years, continuing through 32–41 years in women, should alert physicians to the diagnostic likelihood of polycystic ovary syndrome as a reversible underlying endocrinopathy”, *European Journal of Obstetrics, Gynecology, and Reproductive Biology*, Vol. 122 No. 2, pp. 206–212, doi: 10.1016/J.EJOGRB.2005.03.010.
- Kar, S. (2013), “Anthropometric, clinical, and

- metabolic comparisons of the four Rotterdam PCOS phenotypes: A prospective study of PCOS women”, *Journal of Human Reproductive Sciences*, Vol. 6 No. 3, p. 194, doi: 10.4103/0974-1208.121422.
- Keyif, B. and Yavuzcan, A. (2025), “Visceral and Dysfunctional Adiposity Indices as Predictors of Insulin Resistance and Metabolic Syndrome in Women with Polycystic Ovary Syndrome: A Cross-Sectional Study”, *Medicina* 2025, Vol. 61, No. 3, Page 424, 424, doi: 10.3390/MEDICINA61030424.
- Kim, C.H. and Lee, S.H. (2022), “Effectiveness of Lifestyle Modification in Polycystic Ovary Syndrome Patients with Obesity: A Systematic Review and Meta-Analysis”, *Life*, MDPI, Vol. 12 No. 2, p. 308, doi: 10.3390/LIFE12020308/S1.
- Kim, J.J. and Choi, Y.M. (2013), “Dyslipidemia in women with polycystic ovary syndrome”, *Obstetrics & Gynecology Science*, Vol. 56 No. 3, p. 137, doi: 10.5468/OGS.2013.56.3.137.
- Kumariya, S., Ubba, V., Jha, R.K. and Gayen, J.R. (2021), “Autophagy in ovary and polycystic ovary syndrome: role, dispute and future perspective”, *Autophagy*, Vol. 17 No. 10, pp. 2706–2733, doi: 10.1080/15548627.2021.1938914.
- Legro, R.S., Kusanman, A.R. and Dunaif, A. (2001), “Prevalence and predictors of dyslipidemia in women with polycystic ovary syndrome”, *The American Journal of Medicine*, Vol. 111 No. 8, pp. 607–613, doi: 10.1016/S0002-9343(01)00948-2.
- Marbaniang, G. (2023), “Study of lipid profile in polycystic ovarian syndrome: a case control study in tertiary care hospital”, *International Journal Reprod Contracept Obstet Gynecol*, Vol. 12 No. 12, pp. 3561–3565, doi: 10.18203/2320-1770.Ijrcog.20233634.
- Mirdamadi, A., Riahiinejad, S. and Varnaseri, S. (2020), “The association between anthropometric parameters and cardiovascular risk indicators in women with polycystic ovarian syndrome”, *ARYA Atherosclerosis, Isfahan University of Medical Sciences(IUMS)*, Vol. 16 No. 1, pp. 39–43, doi: 10.22122/ARYA.V16I1.1804.
- Obesity: preventing and managing the global epidemic. Report of a WHO consultation.” (2000), *World Health Organization - Technical Report Series*, Vol. 894.
- Rasool, S. (2021), “Obesity Parameters in Polycystic Ovary Syndrome in Duhok City”, *Journal of Zankoy Sulaimani - Part A, Journal of Zankoy Sulaimani - Part A*, Vol. 23 No. 1, pp. 1–6, doi: 10.17656/JZS.10835.
- Rocha, M.P., Marcondes, J.A.M., Barcellos, C.R.G., Hayashida, S.A.Y., Curi, D.D.G., Da Fonseca, Â.M., Bagnoli, V.R., *et al.* (2011), “Dyslipidemia in women with polycystic ovary syndrome: Incidence, pattern and predictors”, *Gynecological Endocrinology*, Vol. 27 No. 10, pp. 814–819, doi: 10.3109/09513590.2010.508852.
- Rojas, J., Chávez, M., Olivar, L., Rojas, M., Morillo, J., Mejías, J., Calvo, M., *et al.* (2014), “Polycystic ovary syndrome, insulin resistance, and obesity: navigating the pathophysiologic labyrinth”, *International Journal of Reproductive Medicine*, Int J Reprod Med, Vol. 2014, pp. 1–17, doi: 10.1155/2014/719050.
- Akshaya, S., & Bhattacharya, R. (2016), “Comparative study of clinical profile of lean and obese polycystic ovary syndrome women”, *International Journal of Reproduction, Contraception, Obstetrics and Gynecology*, Vol. 5 No. 8, pp. 2530–2533, doi: 10.

- 18203/ 2320- 1770. IJRCOG 20162173.
- Saghafi-asl, M., Pirouzpanah, S. and Ebrahimi-mameghani, M. (2013), "Lipid Profile In Relation To Anthropometric Indices and Insulin Resistance in Overweight Women with Polycystic Ovary Syndrome", Vol. 3 No. 2, pp. 206–216, doi: 10.5681/HPP.2013.024.
- Shen, S.H., Shen, S.Y., Liou, T.H., Hsu, M.I., Chang, Y.C.I., Cheng, C.Y., Hsu, C. Sen, *et al.* (2015), "Obesity and inflammatory biomarkers in women with polycystic ovary syndrome", *European Journal of Obstetrics, Gynecology, and Reproductive Biology, European Journal of Obstetrics, Gynecology, and Reproductive Biology*, Vol. 192, pp. 66–71, doi: 10.1016/J.EJOGRB.2015.06.022.
- Su, P., Chen, C. and Sun, Y. (2025), "Physiopathology of polycystic ovary syndrome in endocrinology, metabolism and inflammation", *Journal of Ovarian Research*, BioMed Central Ltd, Vol. 18 No. 1, pp. 1–10, doi: 10.1186/S13048-025-01621-6/METRICS.
- Talbott, E.O., Guzick, D.S., Sutton-Tyrrell, K., McHugh-Pemu, K.P., Zborowski, J. V., Remsberg, K.E. and Kuller, L.H. (2000), "Evidence for association between polycystic ovary syndrome and premature carotid atherosclerosis in middle-aged women", *Arteriosclerosis, Thrombosis, and Vascular Biology*, Lippincott Williams and Wilkins, Vol. 20 No. 11, pp. 2414–2421, doi: 10.1161/01.ATV.20.11.2414/ASSET/AD0B5F05- 9E04- 45A6-939D-D380BA30D59D/ASSETS/GRAPHIC/HQ1101487001.JPEG.
- Thathapudi, S., Kodati, V., Erukkambattu, J., Katragadda, A., Addepally, U. and Hasan, Q. (2014), "Anthropometric and Biochemical Characteristics of Polycystic Ovarian Syndrome in South Indian Women Using AES-2006 Criteria", *International Journal of Endocrinology and Metabolism*, Vol. 12 No. 1, doi: 10.5812/IJEM.12470.
- Vine, D., Ghosh, M., Wang, T. and Bakal, J. (2023), "Increased Prevalence of Adverse Health Outcomes Across the Lifespan in Those Affected by Polycystic Ovary Syndrome: A Canadian Population Cohort", *CJC Open*, Elsevier Inc., Vol. 6 No. 2Part B, p. 314, doi: 10.1016/J.CJCO.2023.12.010.
- Wild, R.A., Rizzo, M., Clifton, S. and Carmina, E. (2011), "Lipid levels in polycystic ovary syndrome: systematic review and meta-analysis", *Fertility and Sterility*, Vol. 95 No. 3, doi: 10.1016/J.FERTNSTERT.2010.12.027.
- Wu, C.H., Chiu, L.T., Chang, Y.J., Lee, C.I., Lee, M.S., Lee, T.H. and Wei, J.C.C. (2020), "Hypertension Risk in Young Women With Polycystic Ovary Syndrome: A Nationwide Population-Based Cohort Study", *Frontiers in Medicine*, Vol. 7, doi: 10.3389/FMED.2020.574651.
- Zhuang, C., Luo, X., Wang, W., Sun, R., Qi, M. and Yu, J. (2022), "Cardiovascular Risk According to Body Mass Index in Women of Reproductive Age With Polycystic Ovary Syndrome: A Systematic Review and Meta-Analysis", *Frontiers in Cardiovascular Medicine*, Vol. 9, p. 822079, doi: 10.3389/FCVM.2022.822079/BIBTEX