

ORIGINAL ARTICLE

PREVALENCE OF DYSLIPIDAEMIA AMONG PATIENTS OF POLYCYSTIC OVARIAN SYNDROME: A CROSS SECTIONAL ANALYTICAL STUDY

Saleha Zafar, Tabinda Yasmin*, Rabia Saeed**, Javaria Aslam***, Tanzila Rafiq[†], Ammar Bin Saad^{††}

Department of Pathology, Quaid-e-Azam Medical College, Bahawalpur, *Nawab Sir Sadiq Hospital, Bahawalpur, **CMH Institute of Medical Sciences Bahawalpur, ***Department of Medicine, Quaid-e-Azam Medical College, Bahawalpur, [†]Department of Obstetrics and Gynaecology, CMH Institute of Medical Sciences, Bahawalpur, ^{††}Department of Pathology, Ayub Medical College, Abbottabad, Pakistan

Background: Polycystic ovarian disease is a genetically complicated condition, most prevalent in women of reproductive age group. It is strongly associated with obesity, dyslipidaemia, and menstrual disorders. Objective of this study was to determine the prevalence of dyslipidaemia in patients of polycystic ovarian syndrome (PCOS). **Method:** A cross-sectional analytical study was conducted at the Department of Gynaecology, Bahawal Victoria Hospital, Bahawalpur, from 1st Jan to 31st Dec 2021. All newly diagnosed patients of PCOS between the ages of 18 and 35 with a BMI <25 Kg/m² were included in the study. Participants were divided into subgroups according to their age and BMI. The fasting lipid profile was measured through a blood sample taken from each patient after an overnight fast. Total cholesterol >200 mg/dL, LDL-C >130 mg/dL, Triglycerides >150 mg/dL, and HDL-C <40 mg/dL were labelled as dyslipidaemia. Chi-square test was used to calculate and estimate the relative risk (RR) of any associations observed. **Results:** Among total 286 analysed patients of PCOS, mean age was 24.4±5.367 years and mean BMI was 21.01±1.912 Kg/m². Sixty nine (24.13%) patients had dyslipidaemia. Patients in the 18–27 age group and a BMI of 21–23 Kg/m² were more likely to have dyslipidaemia with PCOS, [RR 44.90; 95% CI (16.93, 119.04); *p*<0.001], and [RR 32.12; 95% CI (8.02, 128.61); *p*<0.001] respectively. **Conclusion:** Dyslipidaemia is seen in 24.13% individuals with PCOS. Screening in routine is recommended.

Keywords: Polycystic ovarian syndrome, PCOS, dyslipidaemia, metabolic syndrome

Pak J Physiol 2024;20(1):19–21

INTRODUCTION

Polycystic ovary syndrome (PCOS) is the most prevalent ovarian condition that is associated with androgen excess in women, which explains the immense attention of endocrinologists. This syndrome can result in clinical hyperandrogenism, biological dysovulation, or even infertility. Global incidence of PCOS is 5 to 18% among women of reproductive age.¹ Although the biochemical mechanism behind PCOS pathogenesis stays widely undefined, accumulating data suggests that hyperandrogenism plays an essential role in PCOS development and sequelae. PCOS patients are more likely to develop metabolic syndrome.² There is a considerable risk of cardiovascular disease in PCOS-afflicted women if these risk factors are present.³ Atherogenic dyslipidemia, raised blood pressure, dysglycaemia, a pro-thrombotic condition, and a pro-inflammatory state make up the metabolic syndrome, which causes atherosclerotic cardiovascular disease (ASCVD). Atherogenic dyslipidaemia raises plasma triglycerides, LDL-C and lowers HDL-C.^{4,5} Obesity is a risk factor for both dyslipidaemia and PCOS, hence women with PCOS should be evaluated for dyslipidemia.^{6,7}

Dyslipidaemia, defined as elevated total or low density lipoprotein cholesterol (LDL-C) or low levels of

high density lipoprotein (HDL-C), is an important risk factor for coronary heart disease and stroke.⁸ It can lead to symptomatic coronary artery disease, stroke and peripheral artery disease. High levels of triglycerides (TG) >1,000 mg/dL (>11.3 mmol/L) can cause acute pancreatitis.⁷ Statins are the most frequently used drugs to treat dyslipidaemia. By preventing the liver's ability to produce cholesterol, it lowers LDL levels. Proprotein convertase subtilisin/kexin type 9 (PCSK 9) inhibitors and fibrates are among the non-statin medications.⁹ Lifestyle adjustments may lower cholesterol and TG. The first step is dietary modification; saturated fat, processed carbohydrates, and alcohol should be reduced. Fruits, vegetables, lean proteins, and whole grains may assist.^{9,10}

It is important to diagnose dyslipidaemia in patients with PCOS as early as possible to prevent long term complications of the cardiovascular system. Moreover, early recognition and treatment of dyslipidaemia may also help in the treatment of PCOS.

MATERIAL AND METHODS

After approval from the Ethical Review Board at DME/QAMC Bahawalpur, this cross-sectional analytical study was conducted from 1st Jan 2021 to 31st Dec 2021. Data was collected from patients visiting the

Gynaecology Departments at Bahawal Victoria Hospital, Quaid-e-Azam Medical College, Bahawalpur. Sample size of 300 was calculated with anticipated proportion 24.7%, Confidence Interval 95%, and required precision (d) of 0.05.

All patients in age group of 18–35 years and diagnosed with PCOS on ultrasonography were included in the study after their informed written consent. Demographic information like age and marital status, were noted. Exclusion criteria for the study were those with history of familial hyperlipidaemia, ischemic heart disease, diabetes mellitus, thyroid disease, active smoking, and those taking oral contraceptives, steroids, or lipid-lowering medications. Body Mass Index (BMI) of all participants were noted and those who had BMI >25 Kg/m² or <18 Kg/m² were excluded from the study.

After an overnight fast, 3 mL blood sample was obtained from each patient and analysed in the Pathology Department of QAMC, Bahawalpur. Total cholesterol, HDL-C, LDL-C and Triglycerides were measured on the sample. All tests were performed on a completely automated chemistry analyzer AU-630, and all levels were measured in mg/dL. Total cholesterol >200 mg/dL, LDL-C >130 mg/dL, Triglycerides TG >150 mg/dL, and HDL-C <40 mg/dL were labelled as dyslipidemia.¹¹

The frequency and percentage for the categorical variables were calculated, as were the Mean±SD for the continuous variables. To determine the impact of effect modifiers on the dyslipidaemia, age and BMI stratification was employed. The post-stratification chi-square test was used to determine the relative risk of dyslipidaemia among various age and BMI groups, and $p < 0.05$ was considered statistically significant.

RESULTS

Total 286 patients out of 300 met the inclusion criteria, 210 (73.43%) were from age group 18–27 years. Mean age of the patients was 24.40±5.367 years and mean BMI was 21.01±1.912 Kg/m², [140 (49%) had BMI 18–20 Kg/m² and 146 (51%) had BMI 21–23 Kg/m²]. Sixty-nine (24.13%) patients had dyslipidaemia. Types of dyslipidaemia among these patients are given in Table-1.

When the participants were divided into different age groups and analysed for the presence or absence of dyslipidaemia, it was found that 65 (85.53%) patients from age group 28–35 years had dyslipidemia. Risk of having dyslipidaemia was greater in this age group when compared with patients of age group 18–27 years [65 (85.53%) vs 4 (1.9%); RR 44.90; 95% CI (16.93, 119.04); $p < 0.001$]. (Table-2).

When patients of different BMI groups were analysed for the presence or absence of dyslipidaemia and relative risk of having dyslipidaemia was calculated

among these group, it was found that patients of BMI 21–23 Kg/m² were more likely to have dyslipidaemia as compared to those of BMI 18–20 Kg/m² [67 (46%) vs 2 (1.43%); RR, 32.12; 95%CI (8.02, 128.61); $p < 0.001$]. (Table-3).

Table-1: Types of dyslipidemia in the study group

Type of Dyslipidaemia	Number	Percentage
Total Cholesterol >200 mg/dL	30	43.5
HDL <40 mg/dL	7	10.1
LDL-C >130 mg/dL	5	7.3
Triglycerides >150 mg/dL	27	39.1

Table-2: Age distribution of dyslipidaemia patients

Age Group	Dyslipidemia			p
	Yes (%)	No (%)	Total	
18–27 Year	4 (1.9)	206 (98.1)	210 (73.43)	0.000
28–35 Year	65 (85.53)	11 (14.47)	76 (26.57)	
Total	69 (24.13)	217 (75.87)	286	

Table-3: BMI among dyslipidaemia patients

BMI Group	Dyslipidemia			p
	Yes (%)	No (%)	Total	
18–20 Kg/m ²	2 (1.43)	138 (98.57)	140 (49)	0.000
21–23 Kg/m ²	67 (46)	79 (54)	146 (51)	
Total	69 (24.13)	217 (75.87)	286	

DISCUSSION

Polycystic ovary syndrome is an emerging reproductive disorder in young girls at pubertal age and most commonly associated with dyslipidemias.¹² A study showed that PCOS is strongly associated with obesity and dyslipidaemias in reproductive age group women.¹³ A definitive criteria for diagnosis of hyperlipidaemia is not clearly explained in most of the studies. It is recommended by American College of Obstetricians and Gynaecologists to consider fasting lipid profile for risk assessment of cardiovascular disease in women with PCOS.¹⁴

Obesity, polycystic ovaries, clinical or biochemical hyperandrogenism, and prolonged anovulation are hallmarks of PCOS. Our results provide evidence of association between deranged lipid profile in reproductive age women. However, Joham AE *et al*¹⁵ revealed that oligomenorrhoea or amenorrhoea is linked to hyperandrogenism, and hirsutism or acne may occur as clinical symptoms. Although several pathophysiologic explanations for the emergence of PCOS have been put forth, insulin resistance (IR) is now recognized as being relevant to the disease. Women with PCOS who have IR have an increased risk of type II diabetes and cardiovascular disease.¹⁶

Insulin resistance, hyperinsulinemia, and obesity are often linked with a higher risk of developing metabolic syndrome and type 2 diabetes. The metabolic syndrome is a collection of cardiovascular disease-related risk factors. It is typified by central obesity, high TG, LDL cholesterol levels, and insulin resistance. Moini *et al*¹⁷ found that among women of reproductive

age with PCOS, the prevalence of metabolic syndrome was 22.7%, which was comparable to the prevalence of dyslipidaemia among PCOS patients in this study. Women with PCOS have a high prevalence of metabolic syndrome and its individual components such as low HDL levels.

In a study by Kim JJ *et al*¹⁸, 865 consecutive PCOS patients had a mean age of 24.9, a mean BMI of 22.4 Kg/m², and frequency of dyslipidaemia as 35.7%. Kim JJ *et al*¹⁹ have reported that in 166 women with PCOS, frequency of raised Triglycerides was 26.7% and frequency of low HDL-C was 30%. Hong Y *et al*²⁰, found the prevalence of dyslipidaemia in PCOS women as 24.7%, and it was significantly greater in the insulin resistance group compared to the non-insulin resistance group (39.9% vs 15.4%, $p < 0.05$).

CONCLUSION

Patients with PCOS had a prevalence of dyslipidaemia as 24.13% making them prone to developing metabolic syndrome and hence an increased risk of cardiovascular disease. Patients with polycystic ovarian syndrome should be tested for dyslipidaemia.

REFERENCES

1. Joham AE, Norman RJ, Stener-Victorin E, Legro RS, Franks S, Moran LJ, *et al*. Polycystic ovary syndrome. *Lancet Diabetes Endocrinol* 2022;10:668–80.
2. Abraham Gnanadass S, Divakar Prabhu Y, Valsala Gopalakrishnan A. Association of metabolic and inflammatory markers with polycystic ovarian syndrome (PCOS): an update. *Arch Gynecol Obstet* 2021;303(3):631–43.
3. Osibogun O, Ogunmoroti O, Michos ED. Polycystic ovary syndrome and cardiometabolic risk: Opportunities for cardiovascular disease prevention. *Trends Cardiovasc Med* 2020;30(7):399–404.
4. Grundy SM. Metabolic syndrome update. *Trends Cardiovasc Med* 2016;26(4):364–73.
5. Jatoi NA, Elamin YA, Said AH, Al-Namer B, Al-Muallim FA, Al-Nemer FF, *et al*. Prevalence of cardiovascular risk factors among patients with diabetes mellitus type 2 at King Fahad University Hospital, Saudi Arabia. *Cureus* 2022;14(9):e29489.
6. Legro RS. Obesity and PCOS: Implications for diagnosis and treatment. *Semin Reprod Med* 2012;30(6):496–506.
7. Monteiro R, Azevedo I. Chronic inflammation in obesity and the metabolic syndrome. *Mediators Inflamm* 2010;2010:289645.
8. Jang AY, Han SH, Sohn IS, Oh PC, Koh KK. Lipoprotein(a) and cardiovascular diseases—revisited. *Circ J* 2020;84(6):867–74.
9. Coppinger C, Movahed MR, Azemawah V, Peyton L, Gregory J, Hashemzadah M. Phomprehensive review of PCSK9 inhibitors. *J Cardiovasc Pharmacol Ther* 2022;27:1–14.
10. Rhee EJ, Kim HC, Kim JH, Lee EY, Kim BJ, Kim EM, *et al*. 2018 Guidelines for the management of dyslipidemia. *Korean J Intern Med* 2019;34(4):723–71.
11. He N, Ye H. Exercise and Hyperlipidemia. In: Xiao J. (Ed). *Physical exercise for human health*. Singapore: Singapore; 2020. p.79–90.
12. Sadeghi HM, Adeli I, Calina D, Docea AO, Mousavi T, Daniali M, *et al*. Polycystic ovary syndrome: a comprehensive review of pathogenesis, management, and drug repurposing. *Int J Mol Sci* 2022;23(2):583.
13. Wang C, Wu W, Yang H, Ye Z, Zhao Y, Liu J, *et al*. Mendelian randomization analyses for PCOS: evidence, opportunities, and challenges. *Trends Genet* 2022;38(5):468–82.
14. Tingthanatikul Y, Sripilaipong S, Vallibhakara O, Sophonsritsuk A, Weerakiet S, Vallibhakara SA. A comparative study of LDL-C levels in polycystic ovary syndrome women with different cardiovascular risks according to American Heart Association criteria. *J Med Assoc Thailand* 2017;100(9):927–34.
15. Joham AE, Palomba S, Hart R. Polycystic Ovary Syndrome, obesity, and pregnancy. *Semin Reprod Med* 2016;34(2):93–101.
16. Moghetti P, Tosi F. Insulin resistance and PCOS: chicken or egg? *J Endocrinol Invest* 2021;44(2):233–44.
17. Moini A, Javanmard F, Eslami B, Aletaha N. Prevalence of metabolic syndrome in polycystic ovarian syndrome women in a hospital of Tehran. *Iran J Reprod Med* 2012;10(2):127–30.
18. Kim JJ, Choi YM. Dyslipidemia in women with polycystic ovary syndrome. *Obstet Gynecol Sci* 2013;56(3):137–42.
19. Kim JJ, Hwang KR, Oh SH, Chae SJ, Yoon SH, Choi YM. Prevalence of insulin resistance in Korean women with polycystic ovary syndrome according to various homeostasis model assessment for insulin resistance cutoff values. *Fertil Steril* 2019;112(5):959–66.e1.
20. Hong Y, Yang D, Liu W, Zhao X, Chen X, Li L. Dyslipidemia in relation to body mass index and insulin resistance in Chinese women with polycystic ovary syndrome. *J Biol Regul Homeost Agents* 2011;25(3):365–74.

Address for Correspondence:

Dr Ammar Bin Saad, Assistant Professor Haematology, Department of Pathology, Ayub Medical College Abbottabad, Pakistan. **Cell:** +92-311-4549201

Email: ammarbinsaad1@yahoo.com

Received: 8 Nov 2023

Reviewed: 28 Feb 2024

Accepted: 2 Mar 2024

Contribution of Authors:

SZ: Design of work, Acquisitions and Analysis

TY: Concept and Design of work

RS: Interpretation and Drafting

JA: Final Approval and Agreement to be accountable for all aspects of work

TR: Results tabulation and Interpretation

ABS: Drafting, Acquisitions and Analysis

Conflict of Interest: None

Funding: None