E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/16.01.554

Research Article

Association of Metabolic Syndrome with Non-Alcoholic Fatty Liver Disease in Women Diagnosed With Polycystic Ovary Syndrome: A Multicenter Cross-Sectional Study in Pakistan

Talha Qureshi[1*], Nadeem Bajkani[2], Muhammad Faisal Rashid[3], Muhammad Jaffar[4], Hakro Naveed Ahmed[5], Ghulam Rabani Bugty[6]

[1*]Senior Registrar, Department of Gastroenterology, Dr. Ziauddin Hospital, Sukkur, Sindh, Pakistan.
[2]Assistant Professor, Department of Gastroenterology, Gambat Medical College, Gambat, Sindh, Pakistan.
[3]Assistant Professor, Department of Gastroenterology, Pak Red Crescent Medical and Dental College Hospital, Lahore, Pakistan.
[4]Senior registrar, Department of Medicine, Ziauddin Hospital, Sukkur, Pakistan.
[5]Consultant Gastroenterologist, Department of Gastroenterology, Jinnah Postgraduate Medical Centre, Karachi, Pakistan.
[6]Senior Registrar, Department of Gastroenterology, Gambat Medical College, Gambat, Sindh, Pakistan.
Corresponding Author: Talha Qureshi
Senior Registrar, Department of Gastroenterology, Dr. Ziauddin Hospital, Sukkur, Sindh, Pakistan.
Received: 14.04.26, Revised: 09.05.26, Accepted: 11.06.26

ABSTRACT

Background: The metabolic disturbances usually seen in women with polycystic ovary syndrome include insulin resistance, abdominal adiposity, abnormal lipid levels and impaired glucose regulation. These abnormalities can lead to development of non-alcoholic fatty liver disease. But there is still a lack of evidence in local clinical populations in Pakistan.
Objective: To determine whether metabolic syndrome is linked with non-alcoholic fatty liver disease in women with polycystic ovary syndrome.
Methods: It was a multicentre cross-sectional study conducted at Dr. Ziauddin Hospital, Sukkur and Gambat Medical college, Gambat, Sindh, Pakistan from March 2024 to March 2025. One hundred reproductive-age women (18–40 years old) with confirmed diagnosis of PCOS were recruited sequentially as a sample. Clinical presentation, anthropometric parameters, blood pressure, fasting glucose, serum lipid parameters, hepatic transaminase levels and abdominal ultrasound findings were recorded in the study records of each woman enrolled. Metabolic syndrome was defined based on adult criteria. Diagnosis of NAFLD was confirmed by ultrasonography of the liver revealing fatty infiltration of the liver after excluding other causes of hepatic steatosis.
Results: The prevalence of ultrasound NASH was 44.0% in women and 39.0% of women had metabolic syndrome. Metabolic syndrome was markedly more common in the fatty liver group than in the non-fatty liver group, affecting 61.4% compared with 21.4%, respectively, and the difference was statistically significant ($p<0.001$). Fatty liver women also showed worse metabolic and liver profile with higher BMI, WC, Fasting glucose, TG, BP, ALT, AST and lower fasting HDL cholesterol. In adjusted analysis, the association between metabolic syndrome and fatty liver disease remained independent.
Conclusion: Among Pakistani women with polycystic ovary syndrome, non-alcoholic fatty liver disease showed a strong relationship with metabolic syndrome. These results indicate that liver evaluation and early metabolic screening is warranted in this high-risk population.

Keywords: Polycystic ovary syndrome; metabolic syndrome; non-alcoholic fatty liver disease; insulin resistance; dyslipidemia; Pakistan.

INTRODUCTION

Polycystic ovary syndrome is no longer regarded only as a disorder of ovulation and androgen excess. It is still one of the most common endocrine disorders in women of childbearing age and is not only clinically significant in gynaecology but has been found in many women who have a wide metabolic disorder pattern[1]. The disorder may appear with irregular menstrual cycles, infrequent or absent ovulation, excess androgen activity on clinical examination or laboratory testing, subfertility, acne, hirsutism, and polycystic ovarian appearance on ultrasound[2]. In addition

to these reproductive results, many PCOS women also experience decreased insulin sensitivity, greater stomach fat, abnormal lipid (fats) levels, disturbed glucose regulation, and chronic low-grade inflammation[3]. These abnormalities raise the risk for future metabolic and vascular complications such as type 2 diabetes mellitus, hypertension, cardiovascular disease and metabolic liver damage[4]. For this reason, in 2023 the international evidence-based PCOS guideline advised that patients should be evaluated and managed cardiometabolically as well as using a long term multidisciplinary approach rather than managing PCOS for only the fertility or menstrual concerns alone[5].

Fatty liver disease, without excessive alcohol consumption, is one of the most important causes of chronic liver disease worldwide and is strongly associated with obesity, insulin resistance, dyslipidaemia and type 2 diabetes mellitus[6]. The newer terminology of metabolic dysfunction-associated steatotic liver disease was introduced to place greater emphasis on the metabolic background of hepatic fat accumulation[7]. According to recent European guidance, this condition is characterized by excess triglyceride storage within the liver together with at least one cardiometabolic risk factor[8]. This definition is particularly relevant in PCOS because a large number of affected women develop metabolic risk features early in adult life, sometimes before the appearance of overt diabetes, hypertension, or cardiovascular disease[9].

There is a strong pathophysiological basis for linking PCOS with fatty liver disease, and this relationship has important clinical consequences[10]. Insulin resistance represents a central abnormality in both disorders[11]. Adipose tissue releases more free fatty acids to the bloodstream in this condition of poor tissue responsiveness to insulin. These fatty acids enter the liver and are deposited as triglycerides which contribute to hepatic steatosis[12]. Meanwhile, the liver may also produce more fat via de novo lipogenesis, and fat oxidation and lipid export may be less efficient[13]. This results in intrahepatic fat accumulation, oxidative stress, mitochondrial dysfunction, and inflammatory pathways activation[14]. In women with PCOS, androgen excess also might exacerbate this process by promoting visceral fat, increasing insulin resistance, and inducing metabolic stress in the liver, which could lead to fatty liver disease.[15]

Metabolic syndrome is defined as the presence of a cluster of cardiometabolic risks such as central obesity, high triglycerides, low HDL cholesterol, elevated blood pressure, and impaired fasting glucose[16]. These abnormalities are frequently identified in women with PCOS and may form the clinical bridge between ovarian endocrine dysfunction and hepatic fat deposition[1]. Women with PCOS and metabolic syndrome thus could constitute a subpopulation at risk of non-alcoholic fatty liver disease (NAFLD)[2]. Fatty liver disease has been observed before in PCOS and obesity and insulin resistance are important factors that contribute to this association[3]. Meta-analytic evidence has also shown that PCOS is linked to increased incidence of NAFLD, and body mass index seems to be a modifying factor for this association[4].

The importance of detecting fatty liver disease in PCOS is increased by its silent early course[5]. Many women with hepatic steatosis remain asymptomatic, and liver enzymes may be normal or only mildly raised[6]. Over time, if the metabolic disturbance is not caught early on, NAFLD can evolve to steatohepatitis, fibrosis, cirrhosis and, in some cases, hepatocellular carcinoma (HCC)[7]. In a typical gynecology practice, women with PCOS are likely to be seen for menstrual disturbance, weight concerns, acne, hirsutism, and/or infertility, and liver assessment and metabolic risk evaluation may be overlooked in routine practice[8]. This creates a risk of missed or delayed recognition of fatty liver disease, particularly in healthcare systems where systematic metabolic screening is not routinely available[9].

The rising tide of metabolic disease in Pakistan is attributed to the rise of urbanization, sedentary lifestyle, dietary shifts, obesity and type 2 diabetes mellitus (T2DM)[10]. In particular, women with PCOS may be at risk because their reproductive concerns may tend to be the focus of the clinical visit, while metabolic and hepatic abnormalities may be overlooked[11]. Local data exploring the link between metabolic syndrome and non-alcoholic fatty liver disease in Pakistani women with PCOS are still limited, especially from multicentre clinical settings[12]. Most available local studies have focused mainly on reproductive, hormonal, or ovarian features of PCOS, while liver-related outcomes have been less frequently examined[13].

A multicentre evaluation of this relationship is therefore important for improving screening strategies in Pakistani clinical practice[14].

Detecting metabolic syndrome in women with PCOS may help clinicians identify patients who require liver evaluation, structured lifestyle intervention, and long-term cardiometabolic monitoring[15]. The early diagnosis of NAFLD in this population could additionally decrease the future prevalence of advanced liver disease, diabetes mellitus and cardiovascular complications[16].

The aim of the present study was to explore whether there is any correlation between the presence of metabolic syndrome and non-alcoholic fatty liver disease in women with diagnosed PCOS in a multi-centre population in Pakistan. It also compared anthropometric, clinical, and biochemical profiles of women with and without NAFLD with particular emphasis on central obesity, fasting glucose, lipid abnormalities, blood pressure, and liver enzyme changes.

MATERIALS AND METHODS

The work was designed as a cross-sectional analysis involving two centres in Sindh, Pakistan. The data were collected from Dr. Ziauddin Hospital, Sukkur and Gambat Medical College, Gambat from March 2024 to March 2025. The primary aim was to assess the association of metabolic syndrome with ultrasound detected NAFLD in women with PCOS. The patients were approached on the OPD of gynaecology, medicine and endocrinology in both the institutions and enrolment was done only after applying the eligibility criteria for the study.

The sample studied comprised of 100 women diagnosed with PCOS. The recruitment was done sequentially and not randomly, so that all women who were eligible to participate in the outpatient clinics during the study period were recruited until the desired number of women was achieved. Females were screened when they complained of or had some physical signs of PCOS, including irregular periods, difficulty getting pregnant, acne, increased body hair, weight gain without any other known reason, and other reproductive and metabolic characteristics that indicated PCOS.

Women aged 18-40 years were the only ones considered for inclusion. Accepted clinical and pelvic ultrasound criteria were used to diagnose PCOS. Women were considered diagnosed if they had at least two of the following diagnostic features: infrequent or absent ovulation, clinical or biochemical evidence for androgen excess, and ultrasonographic evidence of polycystic ovarian morphology. Other endocrine disorders that might account for the symptoms were excluded before inclusion in the study. These comprised thyroid disease, hyperprolactinaemia, congenital adrenal hyperplasia, androgen-producing tumours and Cushing syndrome.

Women were excluded if any condition could affect the liver status, metabolic profile or interpretation of fatty liver. Exclusion criteria were pregnancy, breastfeeding, heavy alcohol consumption, hepatitis B or C, known chronic liver diseases, drug-induced liver damage, Wilson disease, haemochromatosis and prior bariatric surgery. Exclusion was also for those who had recently used hepatotoxic drugs, systemic corticosteroid, lipid-lowering drugs, or drugs that are known to substantially alter glucose or lipid metabolism. Records that did not have a clinical or laboratory result, or an ultrasound result, were excluded from the final analysis.

A data sheet was filled out for each participant to organize the data. It involved demographic information, menstrual history, infertility status, acne, hirsutism, other androgen excess markers, family history of diabetes, drug history and relevant co-morbidities. Body composition and central adiposity were measured in all subjects. Body weight was measured in kilograms on a calibrated scale, without shoes and in light clothes. Stadiometer was used to record the Standing height in metres. Then a BMI was calculated from weight and height squared. Waist circumference was determined with a non-elastic tape at the end of quiet expiration at the anatomic level between the lowest rib margin and the iliac crest for the assessment of abdominal obesity.

The blood pressure was taken only after the participant was seated and relaxed for at least 5 minutes. Calibrated sphygmomanometer was used to measure the readings. If re-assessment was necessary, the mean of 2 readings was analysed. After an 8–12 hour overnight fast, fasting venous blood was obtained. Biochemical tests performed comprised fasting plasma glucose, total cholesterol, triglycerides, LDL-C, HDL-C, ALT and AST. All laboratory tests were performed at the individual hospital laboratories by standard laboratory biochemical methods with the internal quality control.

Metabolic syndrome was defined using adult diagnostic criteria. The presence of 3 or more recognised components of the metabolic syndrome was used to classify participants into the metabolic syndrome group. These encompassed central obesity,

hypertriglyceridaemia, low HDL-C, raised blood pressure and increased fasting plasma glucose or previously diagnosed diabetes mellitus. The distribution of each component was documented separately and this was compared between women with NAFLD and women without NAFLD.

Liver assessment was made using abdominal ultrasonography. Examination was done by trained radiologists and the presence of hepatic steatosis was detected if typical sonographic features were observed. These were: increased hepatic brightness relative to renal cortex, decreased intrahepatic vascular markings, posterior acoustic attenuation, and poor diaphragmatic definition if possible. Only after excluding alcohol-related fatty liver and other secondary causes of steatosis was a diagnosis of non-alcoholic fatty liver disease made. The ultrasound results were used to classify women into two groups, those with and those without NAFLD.

The clinical endpoint in this study was the presence of NAFLD on ultrasound in a woman with PCOS. The primary condition studied in relation to the outcome of this was the presence of metabolic syndrome. Other variables, which were included in the analysis, were age, BMI, waist circumference, systolic and diastolic blood pressure, fasting plasma glucose, triglycerides, total cholesterol, LDL-C, HDL-C, ALT, AST, menstrual disturbance, infertility, acne and hirsutism.

All collected data were analyzed in SPSS 27.0. The results were reported in numerical values as mean ± standard deviation and in categorical values as number and percentage. The study population was then split into two groups for comparison based on fatty liver status. Independent-samples t-test was used to compare the differences between the two groups in terms of numerical variables. Stata 15.0 software was used for data analysis; categorical data were analyzed using chi-square test and Fisher's exact test was used when the expected cell count was small. An odds ratio with 95% confidence interval was used to assess the strength of association between metabolic syndrome and NAFLD. A multivariable logistic regression was then performed to assess the independent association of metabolic syndrome with NAFLD after accounting for age, BMI, waist circumference, FPG, triglycerides, and liver enzyme level. Statistically significant results were taken at a p-value of less than 0.05.

The ethical review bodies of the participating hospitals gave their consent prior to enrolment. Written informed consent was obtained from each participant after providing information on the study. The research procedures were in accordance with the Declaration of Helsinki. Confidentiality of participant identities was maintained and data used only for the purposes of this study.

RESULTS

There were 100 women with confirmed PCOS that were analyzed. The participants had a mean age of 27.84 ± 5.41 years and a mean body mass index of 29.18 ± 4.76 kg/m². Fatty liver changes were seen in 44 women, and the frequency of NAFLD in the study group was 44.0% on abdominal ultrasound. The remaining 56 participants had no ultrasonographic evidence of hepatic steatosis. Overall, 39 women (39.0%) were diagnosed with metabolic syndrome.

A comparison between the groups revealed that women with NAFLD were, on average, older than women without NAFLD, but both groups fell in the reproductive age category. Those in the NAFLD group also exhibited a higher BMI and larger waist circumference indicative of adiposity and central fat accumulation. Women with fatty liver were also found to have elevated blood pressure levels (both systolic and diastolic). Laboratory comparison revealed that the NAFLD group had higher levels of fasting plasma glucose, triglycerides, ALTs and ASTs, and lower level of HDL cholesterol. As summarized in Table 1, overall women with PCOS who had NAFLD had worse metabolic and liver enzyme profile as compared to those who did not have NAFLD.

Table 1. Baseline Clinical and Biochemical Characteristics of Women with PCOS According to NAFLD Status

Variable	Overall population n=100	NAFLD present n=44	NAFLD absent n=56	p-value
Age, years	27.84 ± 5.41	29.16 ± 5.22	26.80 ± 5.34	0.029
BMI, kg/m²	29.18 ± 4.76	31.42 ± 4.38	27.42 ± 4.28	<0.001
Waist circumference, cm	91.62 ± 10.84	97.18 ± 9.46	87.25 ± 9.65	<0.001

Systolic BP, mmHg	122.46 ± 12.38	127.34 ± 12.06	118.62 ± 11.21	<0.001
Diastolic BP, mmHg	78.92 ± 8.64	82.14 ± 8.38	76.39 ± 7.92	0.001
Fasting glucose, mg/dL	101.38 ± 18.72	110.64 ± 19.38	94.11 ± 14.92	<0.001
Triglycerides, mg/dL	159.72 ± 46.35	183.26 ± 45.18	141.22 ± 38.71	<0.001
HDL-C, mg/dL	41.86 ± 8.92	37.94 ± 7.65	44.94 ± 8.63	<0.001
LDL-C, mg/dL	119.44 ± 31.26	126.72 ± 32.48	113.72 ± 29.34	0.038
ALT, U/L	39.82 ± 18.14	51.36 ± 18.92	30.75 ± 10.86	<0.001
AST, U/L	33.41 ± 14.67	42.11 ± 15.26	26.57 ± 8.94	<0.001

Individuals who exhibited fatty liver changes on ultrasound were the ones who had metabolic syndrome the most. In NAFLD group, 27 women out of 44 met the criteria for metabolic syndrome (61.4%). Among women without ultrasound evidence of hepatic steatosis, only 12 of 56 cases met the same criteria, giving a frequency of 21.4%. This was statistically significant difference between the two groups. In an item-by-item analysis, the most frequent abnormality was an increase in waist circumference followed by low HDL cholesterol and high triglycerides. Among women, the glucose levels, as well as blood pressure levels, were also relatively elevated in women with NAFLD. These all together indicate that various components of metabolic syndrome were significantly associated with fatty liver disease among women with PCOS as illustrated in Table 2.

Table 2. Frequency of Metabolic Syndrome and Its Components According to NAFLD Status

Variable	NAFLD present n=44	NAFLD absent n=56	p-value
Increased waist circumference	34 (77.3%)	24 (42.9%)	0.001
Raised triglycerides	29 (65.9%)	20 (35.7%)	0.003
Reduced HDL-C	31 (70.5%)	25 (44.6%)	0.010
Elevated blood pressure	18 (40.9%)	11 (19.6%)	0.019
Raised fasting glucose	22 (50.0%)	13 (23.2%)	0.005
Overall metabolic syndrome	27 (61.4%)	12 (21.4%)	<0.001

Logistic regression was used to ascertain the predictive value of metabolic syndrome on NAFLD in the studied population. Similarly, women with metabolic syndrome had significantly higher odds of fatty liver disease in the unadjusted model. The crude odds ratio was 5.82, and the 95% confidence interval ranged from 2.39 to 14.17. The association was still present after adjusting for age, body mass index, waist circumference, fasting plasma glucose, triglycerides, HDL-C and alanine aminotransferase. The adjusted odds ratio was 4.12, with a 95% confidence interval of 1.52 to 11.18. These findings support that metabolic syndrome was an independent determinant of NAFLD as shown in Table 3 among women with PCOS.

Table 3. Logistic Regression Analysis for Factors Associated With NAFLD in Women with PCOS

Variable	Crude OR	95% CI	p-value	Adjusted OR	95% CI	p-value
Metabolic syndrome	5.82	2.39–14.17	<0.001	4.12	1.52–11.18	0.005
Age	1.09	1.01–1.18	0.031	1.05	0.96–1.15	0.267
BMI	1.24	1.12–1.37	<0.001	1.15	1.01–1.31	0.034
Waist circumference	1.11	1.05–1.17	<0.001	1.07	1.01–1.14	0.029
Fasting glucose	1.06	1.03–1.09	<0.001	1.03	1.00–1.06	0.041
Triglycerides	1.03	1.01–1.04	<0.001	1.01	1.00–1.03	0.048
HDL-C	0.89	0.84–0.95	<0.001	0.93	0.87–0.99	0.032
ALT	1.09	1.05–1.14	<0.001	1.06	1.01–1.11	0.018

Overall, these study results suggest that metabolic syndrome and non-alcoholic fatty liver disease go hand in hand in women with polycystic ovary syndrome. Women with fatty liver changes exhibited a significantly worse metabolic profile with a higher rate of abdominal obesity, higher fasting glucose, higher triglycerides, lower high-density lipoprotein cholesterol, higher blood pressure and higher liver enzyme activity. After adjusting

for several other confounding variables, this relationship remained, thus supporting the interpretation that the metabolic syndrome is not just a coincidence in women with PCOS. Rather, it must be considered as a clinically significant alert for the presence of enhanced metabolic risk for the liver in this population.

DISCUSSION

In this multi-centre analysis, women with polycystic ovary syndrome exhibited a distinct overlap of metabolic syndrome and ultrasonographically diagnosed non-alcoholic fatty liver disease[1]. Forty-four percent (44.0%) of the participants were diagnosed with fatty liver and 39 percent (39.0%) with metabolic syndrome[2]. Group comparisons were highly significant with 61.4% of women with NAFLD having metabolic syndrome compared to 21.4% of women without NAFLD[3]. This difference indicates that hepatic steatosis in PCOS is closely related to the combined burden of metabolic risk factors rather than to isolated reproductive dysfunction alone[4].

There were also significant differences between the clinical and biochemical profile of women with and without fatty liver. Participants with NAFLD had greater body mass index, wider waist circumference, higher fasting glucose, increased triglyceride levels, raised blood pressure, and reduced HDL cholesterol[5]. Their ALT and AST levels were also higher, suggesting greater hepatocellular biochemical disturbance in the fatty liver group[6]. The results are consistent with the concept that NAFLD in PCOS is a part of a broader metabolic profile, which includes abdominal obesity, insulin sensitivity, atherogenic dyslipidaemia, and liver enzyme abnormality[7].

A shared mechanism that may explain this relationship is insulin resistance, which is a central abnormality in PCOS, metabolic syndrome, and fatty liver disease[8]. Reduced insulin sensitivity increases the movement of free fatty acids from adipose tissue to the liver, where they are converted into stored triglycerides and contribute to hepatic fat accumulation[9]. This process may be amplified by visceral adiposity and androgen excess, both of which can intensify inflammatory signalling, worsen metabolic stress, and increase hepatic lipid deposition[10]. For this reason, women with PCOS and metabolic syndrome should be regarded as a metabolically vulnerable sub-group that is at increased risk for NAFLD[11].

This association was further supported by the regression analysis. After adjustment for age, body mass index, waist circumference, fasting glucose, triglycerides, HDL cholesterol, and alanine aminotransferase, a relationship between metabolic syndrome and NAFLD remained independent[12]. This indicates that metabolic syndrome is no just a reflection of obesity or a biochemical disorder, but it may be a good clinical tool to identify women with PCOS who need a liver-based evaluation[13].

These results are relevant for routine care in Pakistan, where obesity, sedentary behaviour, type 2 diabetes mellitus, and metabolic disorders are increasing[14]. Medical attention may be sought due to menstrual disturbance, infertility, acne, hirsutism, or weight gain, with the hepatic and metabolic aspects of PCOS potentially not receiving as much attention as the others[15]. Including waist circumference, blood pressure, fasting glucose, lipid profile, liver enzymes, and abdominal ultrasonography in the assessment of high-risk women with PCOS may therefore help detect NAFLD earlier[16].

There are several caveats to consider. Because the study was cross-sectional, it could not establish whether metabolic syndrome preceded the development of NAFLD or appeared alongside it[1]. Diagnosis of fatty liver was done by ultrasonography which is convenient for clinical screening, but less sensitive for mild steatosis and fails to accurately stage fibrosis[2]. Also, dietary intake, physical activity, indices of insulin resistance, and androgen levels were not analyzed, nor was there a direct assessment of fibrosis[3]. Larger longitudinal studies using advanced liver assessment methods are required to confirm these findings and evaluate future metabolic and liver-related outcomes[4].

CONCLUSION

Non-alcoholic fatty liver disease detected by ultrasound was strongly associated with metabolic syndrome in women with polycystic ovary syndrome (PCS). Those affected by fatty liver had a greater cardiometabolic burden, evidenced by greater liver enzyme activity, blood pressure, and abdominal adiposity, as well as abnormal glucose and lipid results. Using relevant clinical and biochemical parameters, metabolic syndrome remained as a risk factor for NAFLD in women.

These results indicate that PCOS care should include attention to liver and metabolic risk, not only reproductive symptoms. Further metabolic and hepatic assessment should be considered in women who have abdominal obesity,

dyslipidaemia, impaired fasting glucose, hypertension or elevated transaminases. By identifying early signs and then making lifestyle changes and controlling risk factors, there is the potential to reduce the progression to liver fibrosis, diabetes, and cardiovascular disease.

Competing Interests: The authors declare that they have no competing interests.
Funding: No external funding was received for this study.
Authors' Contributions: TQ contributed to study conception, study design, data collection, manuscript drafting, and final approval of the manuscript. NB contributed to study supervision, clinical evaluation, data interpretation, and critical revision of the manuscript. MFR contributed to methodology development, literature review, data interpretation, and manuscript revision. WM contributed to patient assessment, clinical data collection, and manuscript review. HNA contributed to clinical interpretation, manuscript review, and intellectual content refinement. GRB contributed to data collection, statistical coordination, manuscript editing, and final review. All authors read and approved the final manuscript.

Acknowledgments: The authors acknowledge the clinical, laboratory, radiology, and administrative staff of Dr. Ziauddin Hospital, Sukkur, and Gambat Medical College, Gambat, Sindh, Pakistan, for their support during data collection and patient evaluation.

REFERENCES

1. European Association for the Study of the Liver, European Association for the Study of Diabetes, European Association for the Study of Obesity. EASL-EASD-EASO Clinical Practice Guidelines on the management of metabolic dysfunction-associated steatotic liver disease. J Hepatol. 2024;81(3):492-542. doi:10.1016/j.jhep.2024.04.031.
2. Bansal SK, Bansal MB. Pathogenesis of MASLD and MASH - role of insulin resistance and lipotoxicity. Aliment Pharmacol Ther. 2024;59 Suppl 1:S10-S22. doi:10.1111/apt.17930.
3. Rahman M, Rahman FT, Mallik MU, Saha J, Rahman MM, Azad KAK. Metabolic dysfunctions in polycystic ovary syndrome. J Medicine. 2024;25(1):68-77. doi:10.3329/jom.v25i1.70703.
4. Teede HJ, Tay CT, Laven JJE, Dokras A, Moran LJ, Piltonen TT, et al. Recommendations from the 2023 International Evidence-based Guideline for the assessment and management of polycystic ovary syndrome. J Clin Endocrinol Metab. 2023;108(10):2447-69. doi:10.1210/clinem/dgad463.
5. Christ JP, Cedars MI. Current guidelines for diagnosing PCOS. Diagnostics (Basel). 2023;13(6):1113. doi:10.3390/diagnostics13061113.
6. Manzano-Nunez R, Rivera-Esteban J, Ortiz-Piedrahita P, Vergara-Moreno E, Osorio-Quintero L, Sanz-Vargas D, et al. Non-alcoholic fatty liver disease in patients with polycystic ovary syndrome: a systematic review, meta-analysis, and meta-regression. J Clin Med. 2023;12(3):856. doi:10.3390/jcm12030856.
7. Zhang H, Wang W, Zhao J, Zhang Y, Liu J, Chen Z, et al. Relationship between body composition, insulin resistance, and hormonal profiles in women with polycystic ovary syndrome. Front Endocrinol (Lausanne). 2022;13:1085656. doi:10.3389/fendo.2022.1085656.
8. Shengir M, Krishnamurthy S, Ghali P, Deschenes M, Wong P, Chen T, et al. Non-alcoholic fatty liver disease in premenopausal women with polycystic ovary syndrome: a systematic review and meta-analysis. JGH Open. 2021;5(4):434-45. doi:10.1002/jgh3.12512.
9. Barrea L, Frias-Toral E, Verde L, Ceriani F, Cucalón G, Garcia-Velasquez E, et al. PCOS and nutritional approaches: differences between lean and obese phenotype. Metabol Open. 2021;12:100123. doi:10.1016/j.metop.2021.100123.
10. Bulsara J, Patel P, Soni A, Acharya S. A review: brief insight into polycystic ovarian syndrome. Endocr Metab Sci. 2021;3:100085. doi:10.1016/j.endmts.2021.100085.
11. Cooney LG, Dokras A. Beyond fertility: polycystic ovary syndrome and long-term health. Fertil Steril. 2018;110(5):794-809. doi:10.1016/j.fertnstert.2018.08.021.
12. Friedman SL, Neuschwander-Tetri BA, Rinella M, Sanyal AJ. Mechanisms of NAFLD development and therapeutic strategies. Nat Med. 2018;24(7):908-22. doi:10.1038/s41591-018-0104-9.
13. Wolf WM, Wattick RA, Kinkade ON, Olfert MD. Geographical prevalence of polycystic ovary syndrome as determined by region and race/ethnicity. Int J Environ

Res Public Health. 2018;15(11):2589. doi:10.3390/ijerph15112589.

14. Wu J, Yao XY, Shi RX, Liu SF, Wang XY. A potential link between polycystic ovary syndrome and non-alcoholic fatty liver disease: an update meta-analysis. Reprod Health. 2018;15(1):77. doi:10.1186/s12978-018-0519-2.
15. Targher G, Rossini M, Lonardo A. Evidence that non-alcoholic fatty liver disease and polycystic ovary syndrome are associated by necessity rather than chance: a novel hepato-ovarian axis? Endocrine. 2016;51(2):211-21. doi:10.1007/s12020-015-0640-8.
16. Romanowski MD, Parolin MB, Freitas ACT, Piazza MJ, Basso J, Urbanetz AA. Prevalence of non-alcoholic fatty liver disease in women with polycystic ovary syndrome and its correlation with metabolic syndrome. Arq Gastroenterol. 2015;52(2):117-23. doi:10.1590/S0004-28032015000200008.
17. Alberti KGMM, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation. 2009;120(16):1640-5. doi:10.1161/CIRCULATIONAHA.109.192644.