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Metabolic and Hormonal Predictors of Non-Alcoholic Fatty Liver Disease in PCOS/PMOS: A Clinicobiochemical Study

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ABSTRACT

Introduction: PCOS, newly named PMOS (polyendocrine-metabolic-ovarian-syndrome) is now closely linked to NAFLD fueled by a toxic trifecta of insulin resistance, hormonal shifts and lifestyle choices, which frequently remains underdiagnosed among young PCOS patients in India. This study henceforth assessed the association between fasting insulin levels and ultrasonographically detected fatty liver in young Indian women with PCOS, alongside metabolic and dietary predictions.
Methods: A hospital-based cross-sectional study was conducted among 384 women (aged 18-40) diagnosed with PCOS (Rotterdam criteria). Clinical, biochemical, anthropometric and dietary data were collected. Abdominal ultrasonography was used to detect the grade-level fatty liver. Statistical analysis included chi square tests, t-tests and logistic regression, with $p<0.05$ considered significant.
Results: Fatty liver was present in 62.8% of participants (with PCOS). Elevated fasting insulin (>20 µIU/mL) showed a strong association with fatty liver ($p<0.001$). Women with fatty liver demonstrated significantly higher HOMA-IR, LH/FSH ratio and serum compared to those without fatty liver (all $p<0.001$). High-glycemic diet, fasting insulin, BMI >25 kg/m² WHR >0.85 and high CI were identified as significant independent biomarkers of fatty liver as well as visceral/central obesity. Increased ALT:AST ratio, elevated-TG and decreased-SHBG were identified as significant biomarkers of metabolic-hormonal disturbance, termed PMOS.
Conclusion: This study highlights a high prevalence of fatty liver and metabolic–hormonal disturbances in PCOS/PMOS, primarily mediated by insulin resistance, hyperandrogenism and unhealthy dietary patterns. The findings underscore the necessity of early metabolic screening and lifestyle interventions to mitigate long-term reproductive and metabolic risks.

Keywords: PCOS, PCOM, NAFLD, Insulin Resistance, LH/FSH Ratio, Hyperandrogenism, High-Glycemic Diet, Fatty Liver

INTRODUCTION

Polycystic Ovarian Syndrome (PCOS) represents a multifactorial endocrine-metabolic disorder that has garnered increasing attention over the past few decades due to its rising incidence and complex clinical spectrum. [1] It affects approximately 8% to 13% of women of reproductive age worldwide, with even higher rates in South Asian countries like India, where lifestyle transitions, urbanization, and nutritional shifts have contributed to a surge in metabolic disorders among young women. Traditionally perceived as a gynecological condition due to its hallmark features chronic anovulation, menstrual irregularities, hirsutism, acne and infertility PCOS is now understood as a systemic disorder with significant metabolic implications [2]. Central to its pathophysiology is insulin resistance (IR), an underlying mechanism present in up to 70% of PCOS patients [3], even in those who are not obese. IR promotes compensatory hyperinsulinemia, which not only exacerbates ovarian androgen production but also leads to a cascade of metabolic disruptions that predispose these women to long-term complications including type 2 diabetes mellitus (T2DM), dyslipidemia, cardiovascular disease, and more recently, non-alcoholic fatty liver disease (NAFLD). [4]

NAFLD, defined as hepatic steatosis in the absence of significant alcohol consumption, has emerged as a major public health concern, affecting nearly 25% of the global population and up to 55–70% of women with PCOS [5]. This high co-occurrence is not coincidental; rather, it highlights the shared metabolic roots of both conditions. The liver plays a central role in glucose and lipid metabolism, and the state of chronic hyperinsulinemia seen in PCOS disrupts normal hepatic insulin signaling, leading to triglyceride accumulation within hepatocytes. [6] Furthermore, gonadotropin imbalances, particularly a persistently elevated Luteinizing Hormone to Follicle Stimulating Hormone (LH/FSH) ratio, disrupt the hypothalamic-pituitary-ovarian (HPO) axis and impair follicular maturation, compounding reproductive dysfunction. In addition, hyperandrogenism, another cardinal feature of PCOS, is closely tied to both insulin resistance and hepatic fat accumulation.

Recent studies suggest that the LH/FSH ratio, serum testosterone levels, Body Mass Index (BMI), and dietary habits, particularly the intake of high glycemic index foods, may all play synergistic roles in the development of fatty liver in PCOS patients. [6] In clinical practice, this confluence of reproductive and metabolic abnormalities presents a significant diagnostic and therapeutic challenge. The presence of NAFLD in a young woman with PCOS not only predicts more severe insulin resistance and a higher likelihood of developing overt diabetes, but also serves as a red flag for the possibility of progressive liver disease, including steatohepatitis and cirrhosis, if left unaddressed. [7]

Despite increasing recognition of this interrelationship, studies specifically examining the association between fasting insulin levels, LH/FSH ratio, serum androgens, and fatty liver in PCOS patients remain limited in the Indian subcontinent. [5] Furthermore, population-specific dietary and lifestyle patterns, socioeconomic disparities, and lack of early screening often lead to underdiagnosis and mismanagement of these coexisting conditions. Therefore, there is an urgent need to identify and analyze key hormonal and metabolic indicators that can predict the development of fatty liver in PCOS patients at an early stage. [4,6]In this context, the present study aims to investigate the association between fasting insulin levels, gonadotropin ratios, serum testosterone, body mass index, and dietary factors with the presence of fatty liver in women diagnosed with PCOS. By uncovering these associations, the study endeavors to provide valuable insights for early diagnosis, comprehensive risk assessment, and integrated management strategies for this high-risk population,

thereby preventing long-term complications and improving overall reproductive and metabolic health outcomes. [7]

PCOS is a complex endocrine metabolic disorder with implications extending beyond infertility to include NAFLD. Both conditions share common pathophysiological pathways such as insulin resistance, hyperinsulinemia, obesity, androgen excess and chronic inflammation. NAFLD frequently remains underdiagnosed in young women with PCOS due to limited metabolic and hepatic screening. [5] This study focuses on identifying key hormonal and metabolic predictors fasting insulin, LH/FSH ratio, serum testosterone, BMI, conicity index and dietary patterns associated with fatty liver in PCOS patients.

The study aims to evaluate the association between fasting insulin levels and the presence of fatty liver in patients with Polycystic Ovarian Disease (PCOS), with a focus on determining whether elevated fasting insulin acts as a significant predictor of hepatic steatosis. [7] It also seeks to analyze the distribution of the LH/FSH ratio among PCOS patients with and without fatty liver and to assess its correlation with liver involvement. Additionally, the study compares key hormonal and metabolic parameters, including HOMA-IR, serum testosterone, and body mass index (BMI), conicity index (CI: waist circumference / 0.109 × √(weight/height)) between these groups. The influence of high-glycemic dietary patterns on the risk of fatty liver in PCOS individuals is further evaluated. Finally, logistic regression analysis is employed to identify independent predictors of fatty liver in PCOS, supporting early detection and improved management of high-risk patients. [8]

METHODS

Study Design

This hospital-based cross-sectional observational study was undertaken to explore the frequency and association of fatty liver changes detected by ultrasonography (USG) among patients diagnosed with polycystic ovarian disease (PCOS). A total of 384 female patients were included in the study, all of whom were diagnosed with PCOS based on the widely accepted Rotterdam criteria, which requires at least two out of three features: oligo/anovulation, clinical or biochemical signs of hyperandrogenism, and polycystic ovarian morphology on ultrasound. The study was conducted over a specified period in the gynecology and radiology departments of a tertiary care teaching hospital, ensuring a representative and clinically relevant sample.

Participants

Eligible participants were females aged between 18 and 40 years who presented to the outpatient department with symptoms suggestive of PCOS such as irregular menstrual cycles, hirsutism, acne, or infertility.

Anthropometric and Biochemical measurements & Clinical Outcomes

After obtaining written informed consent, detailed demographic data, clinical history, anthropometric measurements (including BMI, waist-to-hip ratio (WHR) and conicity index (CI: waist circumference / 0.109 × √weight/height)) and laboratory results (where available) were recorded using a structured, pre-tested data collection tool. Each participant underwent a transabdominal pelvic ultrasound for PCOS confirmation and an abdominal ultrasound to screen for hepatic steatosis (fatty liver). The USG examinations were performed by experienced radiologists using standardized imaging protocols. Fatty liver grading was done based on echogenicity patterns as per conventional radiologic criteria. [9]

To maintain internal validity, women with pre-existing liver conditions such as chronic viral hepatitis, autoimmune liver disease, or cirrhosis were excluded. Additionally, participants with a history of significant alcohol intake (more than 20 grams per day), those taking hepatotoxic drugs, or diagnosed with metabolic disorders unrelated to PCOS

(e.g., Wilson's disease) were excluded to avoid confounding variables. [10]

Fasting blood samples were collected after an overnight fast (minimum of 8-12 hours). Serum levels of fasting insulin, serum testosterone, luteinizing hormone (LH), follicle-stimulating hormone (FSH) and sex hormone-binding globulin (SHBG) were quantified using on an automated analyzer. The LH/FSH ratio was subsequently calculated.

Glucose homeostasis and insulin resistance were evaluated using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) calculated via the standard formula:

$$\text{HOMA} - \text{IR} = \frac{\text{Fasting insulin } (\mu\text{U/mL}) \times \text{Fasting glucose } (\text{mg/dL})}{405}$$

To evaluate liver involvement and metabolic status, liver function enzymes including alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were measured. The ratio of ALT:AST was calculated subsequently. Serum triglyceride (TG) was analyzed using an automated enzymatic colorimetric method to assess the lipid profile. All assays were performed in accordance with standard laboratory quality control protocols.

Statistical Analysis

The collected data were entered into Microsoft Excel and analyzed using SPSS (Statistical Package for the Social Sciences) version 19. Descriptive statistics such as frequencies, means, and standard deviations were used to summarize the demographic and clinical characteristics of the participants. The prevalence of fatty liver among PCOS patients was calculated, and inferential statistics including the chi-square test were applied to examine the association between categorical variables such as presence of fatty liver and BMI category, age group, and menstrual irregularity. A p-value of <0.05 was considered statistically significant. The study was approved by the Institutional Ethics Committee, and all procedures were conducted in accordance with the Declaration of Helsinki.

The study was approved by the Institutional Ethics Committee (IEC No.: BMC/IEC/219/25 dated on 24/11/2025) and all procedures were conducted in accordance with the declaration of Institutional Ethics Committee, Burdwan Medical College, West Bengal, India.

RESULTS

Socio-demographic Characteristics

A total of 384 female patients diagnosed with polycystic ovarian syndrome (PCOS) were included in this hospital-based cross-sectional study. The majority of participants (61.2%) were in the age group of 21–30 years, followed by 18–20 years (25.3%), and 31–40 years (13.5%). Most participants were unmarried (67.7%) and urban residents (58.1%). Regarding educational background, 39.8% had completed graduation, 35.4% were educated up to higher secondary level, and the remaining had either postgraduate qualifications or studied up to secondary level. Socioeconomic classification (based on modified BG Prasad scale) showed that 43% belonged to the upper-middle class, 31% to the lower-middle class, and 26% to the lower class.

Hormonal and Metabolic Findings

The comparison of hormonal, metabolic, and central obesity parameters between PCOS women with fatty liver and those without fatty liver demonstrated highly significant differences across all measured variables (Table 1). Fasting insulin levels were markedly elevated in the fatty liver group (19.7 ± 6.4 µIU/mL) compared to the non-fatty liver group (10.3 ± 3.9 µIU/mL), with a highly significant t-value of 15.28 ($p < 0.001$), indicating pronounced hyperinsulinemia. Consistently, HOMA-IR values were significantly higher in the fatty

liver group (4.52 ± 1.23 vs. 2.16 ± 0.94; t = 13.92, p < 0.001), confirming severe insulin resistance in these patients.

Hormonal parameters also showed a significant derangement, with the LH/FSH ratio being elevated in the fatty liver group (2.14 ± 0.65) compared to controls (1.27 ± 0.41), yielding a t-value of 11.74 (p < 0.001). Similarly, serum testosterone levels were significantly higher in the fatty liver group (78.4 ± 18.2 ng/dL vs. 49.6 ± 11.7 ng/dL), with a t-value of 16.56 (p < 0.001), reflecting pronounced hyperandrogenism associated with fatty liver in PCOS patients.

Measures of central obesity were also significantly increased in the fatty liver group. The waist–hip ratio was higher (0.93 ± 0.06 vs. 0.84 ± 0.05; t = 10.21, p < 0.001), and the conicity index was similarly elevated (1.32 ± 0.12 vs. 1.18 ± 0.09; t = 9.85, p < 0.001), indicating greater abdominal fat accumulation and increased cardiometabolic risk.

Liver-related and lipid parameters further strengthened these findings. The ALT:AST ratio was significantly higher in the fatty liver group (1.62 ± 0.41) compared to the non-fatty liver group (1.08 ± 0.29), with a t-value of 14.03 (p < 0.001), suggesting hepatic dysfunction and early features of non-alcoholic fatty liver disease. Triglyceride levels were also significantly elevated in the fatty liver group (186 ± 52 mg/dL vs. 128 ± 37 mg/dL; t = 12.87, p < 0.001), indicating dyslipidemia and metabolic disturbance.

In contrast, SHBG levels were significantly reduced in the fatty liver group (19.6 ± 7.8 nmol/L) compared to the non-fatty liver group (38.4 ± 10.2 nmol/L), with a t-value of 13.95 (p < 0.001), reflecting increased free androgen availability and worsening hyperandrogenic state.

Overall, the findings demonstrate a strong interrelationship between metabolic dysfunction, hyperandrogenism, and abdominal obesity in the development of fatty liver among PCOS patients. The statistically significant differences across all studied parameters highlight the importance of early metabolic assessment, lifestyle modification, dietary regulation, and weight management to reduce the risk of long-term complications such as Type 2 Diabetes Mellitus, cardiovascular disease, and progressive liver dysfunction.

Table 1: Mean Hormonal, Metabolic, and Central Obesity Profile (with Conicity Index and Waist–Hip Ratio)

Parameter	Fatty Liver (n=241) Mean ± SD	No Fatty Liver (n=143) Mean ± SD	t-value	p-value
Fasting Insulin (µIU/mL)	19.7 ± 6.4	10.3 ± 3.9	15.28	<0.001*
HOMA-IR	4.52 ± 1.23	2.16 ± 0.94	13.92	<0.001*
LH/FSH Ratio	2.14 ± 0.65	1.27 ± 0.41	11.74	<0.001*
Serum Testosterone (ng/dL)	78.4 ± 18.2	49.6 ± 11.7	16.56	<0.001*
Waist–Hip Ratio	0.93 ± 0.06	0.84 ± 0.05	10.21	<0.001*
Conicity Index	1.32 ± 0.12	1.18 ± 0.09	9.85	<0.001*

*Significant at p < 0.05

Physiological and Metabolic Findings

The combined analysis of dietary, metabolic, and hormonal factors (Table 2) revealed a highly significant association with fatty liver among PCOS patients. A greater proportion of women consuming a high glycemic diet were found to have fatty liver (78.2%) compared to those following a balanced diet (48.5%), indicating that unhealthy dietary habits may contribute substantially to hepatic fat accumulation and metabolic imbalance.

With respect to insulin status, patients with elevated fasting insulin levels (>20 µIU/mL) demonstrated the highest prevalence of fatty liver (42.7%), whereas the majority of individuals without fatty liver had normal fasting insulin levels (57.3%). The association between fasting insulin and fatty

liver was highly significant ($\chi^2 = 78.65$, $p < 0.001$), supporting the critical role of hyperinsulinemia and insulin resistance in the pathogenesis of fatty liver among women with PCOS.

Similarly, hormonal imbalance assessed through the LH/FSH ratio showed a strong association with fatty liver. An elevated LH/FSH ratio (>2.0) was observed in 41.9% of patients with fatty liver compared to only 9.1% of those without fatty liver, while normal LH/FSH ratios were predominantly found among individuals without fatty liver (64.3%). This association was statistically significant ($\chi^2 = 50.21$, $p < 0.001$), highlighting the contribution of endocrine dysfunction to both reproductive and hepatic abnormalities in PCOS patients.

Overall, these findings indicate a strong interplay between unhealthy dietary patterns, insulin resistance, and hormonal dysregulation in the development of fatty liver among women with PCOS. The statistically significant associations underscore the multifactorial nature of fatty liver disease and the importance of integrated metabolic and hormonal management strategies.

Table 2: Association of Dietary Pattern, Fasting Insulin, and LH/FSH Ratio with Fatty Liver in PCOD Patients (n = 384)

Variables	Categories	Fatty Liver Present (n=241)	Fatty Liver Absent (n=143)	Total	χ^2 value	p-value
Dietary Pattern	High Glycemic Diet	145 (78.2%)	41 (21.8%)	186	—	<0.001*
	Balanced Diet	96 (48.5%)	102 (51.5%)	198		
Fasting Insulin (µIU/mL)	<10 (Normal)	36 (14.9%)	82 (57.3%)	118	78.65	<0.001*
	10–20 (Moderately High)	102 (42.3%)	48 (33.6%)	150		
	>20 (High)	103 (42.7%)	13 (9.1%)	116		
LH/FSH Ratio	<1.5 (Normal)	61 (25.3%)	92 (64.3%)	153	50.21	<0.001*
	1.5–2.0 (Mildly High)	79 (32.8%)	38 (26.6%)	117		
	>2.0 (Elevated)	101 (41.9%)	13 (9.1%)	114		

(*Significant at $p < 0.05$)

Logistic Regression Analysis for Predictors of Fatty Liver in PCOS Patients

Binary logistic regression analysis was performed to identify independent predictors associated with fatty liver among PCOS patients (Table 3). The results demonstrated that several metabolic, dietary, hormonal, and obesity-related factors significantly increased the likelihood of fatty liver development.

A high glycemic diet emerged as a significant independent predictor, with affected individuals showing nearly threefold higher odds of fatty liver (Adjusted OR = 2.91; 95% CI: 1.89–4.52; $p < 0.001$). Elevated fasting insulin levels (>20 µIU/mL) represented the strongest predictor, increasing the odds of fatty liver by approximately 3.87 times (95% CI: 2.31–6.48; $p < 0.001$), thereby reinforcing the major contribution of insulin resistance to hepatic steatosis in PCOS patients.

Hormonal imbalance also significantly influenced fatty liver risk. Patients with an LH/FSH ratio greater than 2 had 2.43 times higher odds of developing fatty liver (95% CI: 1.48–4.01; $p < 0.001$). Additionally, generalized obesity, represented by BMI >25 kg/m², significantly increased the likelihood of fatty liver (Adjusted OR = 2.14; 95% CI: 1.32–3.47; $p = 0.002$).

Measures of central obesity independently predicted fatty liver risk as well. A high Conicity Index was associated with 2.76 times greater odds of fatty liver (95% CI: 1.68–4.55; $p < 0.001$), while a Waist–Hip Ratio >0.85 increased the odds by 2.58 times

(95% CI: 1.57–4.23; p < 0.001). These findings emphasize the important role of abdominal adiposity and altered fat distribution in the pathogenesis of hepatic steatosis among women with PCOS.

Overall, the regression analysis highlights that insulin resistance, hormonal imbalance, central obesity, generalized obesity, and unhealthy dietary habits are all significant independent predictors of fatty liver in PCOS patients. The consistent statistical significance across these variables demonstrates the multifactorial relationship between metabolic dysfunction and hepatic steatosis, emphasizing the need for comprehensive metabolic, nutritional, and endocrine interventions.

Table 3: Logistic Regression Analysis for Predictors of Fatty Liver in PCOD Patients

Variable	Adjusted OR	95% CI	p-value
High Glycemic Diet	2.91	1.89–4.52	<0.001
Fasting Insulin >20 µIU/mL	3.87	2.31–6.48	<0.001
LH/FSH Ratio >2	2.43	1.48–4.01	<0.001
BMI >25 kg/m²	2.14	1.32–3.47	0.002
High Conicity Index	2.76	1.68–4.55	<0.001
Waist–Hip Ratio >0.85	2.58	1.57–4.23	<0.001

DISCUSSION

Polycystic Ovarian Syndrome (PCOS), now increasingly recognized as Polyendocrine metabolic ovarian syndrome (PMOS) [11], is far more than a reproductive disorder; it is a profound endocrine–metabolic disease intricately interwoven with Nonalcoholic Fatty Liver Disease (NAFLD). [11] At the heart of this pathological alliance lies insulin resistance, the master defect driving hormonal disruption, metabolic instability, and progressive hepatic injury. [12] When peripheral tissues fail to respond adequately to insulin, the pancreas compensates through persistent hyperinsulinemia, creating a metabolic environment that fuels both ovarian and hepatic dysfunction simultaneously. [13,14] Excess insulin overstimulates ovarian theca cells, triggering excessive androgen synthesis and intensifying hyperandrogenism, while concurrently suppressing hepatic production of sex hormone-binding globulin (SHBG), thereby increasing circulating free androgens and amplifying endocrine imbalance. [15] This hormonal chaos is further reinforced by an elevated luteinizing hormone (LH) to follicle-stimulating hormone (FSH) ratio, a hallmark abnormality that perpetuates anovulation, follicular arrest, and ovarian dysfunction.

Beyond the ovaries, insulin resistance unleashes widespread metabolic devastation. Enhanced adipose tissue lipolysis floods the circulation with free fatty acids (FFAs), which are rapidly transported to the liver, where they drive excessive triglyceride synthesis and lipid accumulation. This cascade culminates in hepatic steatosis, accompanied by atherogenic dyslipidemia characterized by elevated very low-density lipoprotein (VLDL) and low-density lipoprotein (LDL) levels alongside reduced high-density lipoprotein (HDL) concentrations[15]. Dietary indiscretions, particularly chronic intake of high-glycemic foods, intensify this pathological cycle by provoking recurrent glucose surges and hyperinsulinemia, thereby worsening obesity, insulin resistance, dyslipidemia, and hepatic fat deposition. Consequently, NAFLD emerges not merely as a hepatic complication of PMOS/PCOS, but as a direct metabolic extension of the syndrome itself.

As fat progressively accumulates within hepatocytes, oxidative stress, mitochondrial dysfunction, and inflammatory signaling pathways become activated, transforming simple steatosis into inflammatory steatohepatitis with the potential for fibrosis and irreversible liver injury. Hyperandrogenism further aggravates this process by promoting visceral adiposity and

central fat redistribution, particularly abdominal obesity, which acts as a metabolically active inflammatory reservoir. [15] Adipokines and pro-inflammatory cytokines released from visceral fat intensify insulin resistance and sustain chronic low-grade systemic inflammation, establishing a vicious self-perpetuating cycle of endocrine and metabolic deterioration. [14,15] Clinically, this interplay manifests through elevated alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels, with ALT emerging as an early biochemical sentinel of metabolic liver injury. [13] Simultaneously, fasting hyperglycemia, hyperinsulinemia, adverse lipid profiles, and elevated insulin resistance indices serve as powerful predictors of NAFLD severity in women with PMOS/PCOS. [13,14]

Anthropometric markers provide additional insight into disease burden. Although body mass index (BMI) reflects generalized obesity, waist–hip ratio (WHR) and conicity index more accurately capture visceral adiposity and central obesity, rendering them superior predictors of insulin resistance, cardiometabolic dysfunction, and fatty liver progression [16]. Thus, the convergence of insulin resistance, dyslipidemia, hyperandrogenism, gonadotropin imbalance, reduced SHBG levels, poor dietary habits, and visceral fat accumulation orchestrates a multifaceted pathogenic network that accelerates NAFLD progression in PMOS/PCOS patients. [17]

The clinical consequences of PMOS/PCOS extend far beyond metabolic dysfunction. Reproductive impairment remains a defining challenge, with chronic anovulation contributing to infertility in nearly half of affected women. Obesity and insulin resistance further compromise reproductive outcomes by increasing miscarriage rates and reducing live birth success. Pregnancy itself becomes high risk, with significantly increased incidence of gestational diabetes mellitus, preeclampsia, pregnancy-induced hypertension, and preterm delivery. Simultaneously, long-term cardiometabolic complications emerge as major threats, including hypertension, metabolic syndrome, type 2 diabetes mellitus, endothelial dysfunction, subclinical atherosclerosis, and increased cardiovascular morbidity. Biomarkers such as elevated C-reactive protein (CRP), homocysteine, carotid intima-media thickness (CIMT), and coronary artery calcification (CAC) underscore the silent but progressive cardiovascular burden carried by these women. [18]

Moreover, chronic anovulation exposes the endometrium to prolonged unopposed estrogen stimulation, substantially increasing the risk of endometrial hyperplasia and carcinoma [19]. The syndrome also imposes profound psychosocial distress. Hirsutism, acne, obesity, infertility, and body image dissatisfaction frequently precipitate anxiety, depression, social withdrawal, and diminished quality of life. Alarmingly, delayed recognition and inconsistent diagnostic criteria leave a substantial proportion of cases undiagnosed, allowing metabolic and hepatic complications to silently progress for years.

Despite advances in understanding its pathophysiology, PMOS/PCOS remains a lifelong therapeutic challenge without a definitive cure [11,20,21]. Lifestyle modification continues to serve as the cornerstone of management, yet sustainable metabolic improvement often requires prolonged commitment and multidisciplinary intervention. [22] Pharmacological therapies such as metformin, ovulation-inducing agents, hormonal regulators, and insulin-sensitizing drugs [3,23,24] may alleviate symptoms and improve metabolic outcomes, but they seldom eliminate the underlying disorder. Similarly, NAFLD management [8] remains largely dependent on weight reduction and metabolic correction due to the absence of universally effective targeted therapies. Therefore, early diagnosis, aggressive metabolic screening, individualized hormonal assessment, and integrated multidisciplinary care are indispensable to preventing the progressive hepatic, reproductive, cardiovascular, and

psychological consequences associated with PMOS/PCOS.

CONCLUSION

This cross-sectional study accomplishes that fatty liver disease is highly prevalent among women with PCOS, sturdily associated with insulin resistance, hormonal imbalance and unhealthy dietary patterns. Hyperinsulinemia plays a central role in linking hepatic steatosis with ovarian dysfunction by promoting androgen excess and altered gonadotropin secretion. These findings designate that PCOS and fatty liver share a common metabolic pathway. Early metabolic screening and targeted lifestyle modifications highlight the need for a comprehensive approach to reduce long-term reproductive and metabolic complications in this population.

Declaration

Ethical Approval: This study involving human participants was planned, conducted, and reported in accordance with the ethical principles of the World Medical Association Declaration of Helsinki.

Ethical approval for this study was obtained from the Institutional Ethics Committee, Burdwan Medical College, West Bengal, India (Approval No. BMC/IEC/79/2025; Date: 19.05.2025).

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Informed Consent Statement: Written informed consent was obtained from all participants prior to participation in the study.

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Authors' Contribution:

Dr. Sudip Ghosh contributed to statistical analysis and interpretation of data. Ms. Ritika Seth and Mr. Suman Das contributed to data collection, literature review and manuscript preparation. Mrs. Pallabi Chatterjee helped in biochemical assays and interpretation of data. Dr. Sarmishtha Chatterjee contributed to study conception, supervision, manuscript drafting and correspondence with the journal. All authors reviewed and approved the final manuscript according to ICMJE authorship criteria.

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