



Prevalence of Metabolic Dysfunction-Associated Steatotic Liver Disease in Women with Polycystic Ovary Syndrome and its Association with PCOS Phenotypes

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Abstract

Background: Polyendocrine Metabolic Ovarian Syndrome (PMOS) is recognized as the most prevalent endocrine disorder among women of reproductive age, with an estimated prevalence of approximately 18% according to current Rotterdam diagnostic criteria. Conditions such as dyslipidemia, insulin resistance, hyperandrogenism, central adiposity, and chronic inflammation are frequently observed in women diagnosed with PMOS. These conditions predispose such individuals to an increased risk of developing Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), which is the most prevalent chronic liver disease worldwide, affecting approximately 25% of the global population. PMOS and MASLD represent two interconnected metabolic disorders that share overlapping risk factors

Objective: To determine the prevalence of MASLD in women with PCOS and correlate it with PCOS phenotypes.

Methods: This cross-sectional observational study was conducted in the Department of Obstetrics and Gynaecology, Jawaharlal Nehru Medical College, Aligarh Muslim University (A.M.U.), after obtaining approval from the Institutional Ethics Committee. A total of 50 women aged 18–40 years diagnosed with polycystic ovary syndrome (PCOS) according to the Rotterdam 2003 criteria were consecutively enrolled after obtaining written informed consent

Results: MASLD prevalence significantly higher in PCOS (32%) vs controls (4%), $p < 0.001$. Phenotype A (HA+OD+PCOM) was the most common (72%) hyperandrogenic phenotypes (A, B, C) show the highest fatty liver rates

Conclusions: MASLD prevalence significantly higher in PCOS as compared to non PMOS

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Introduction

Polyendocrine Metabolic Ovarian Syndrome (PMOS), formerly known as Polycystic Ovary Syndrome (PCOS), and Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), previously referred to as Non-Alcoholic Fatty Liver Disease (NAFLD), are two interconnected metabolic disorders characterized by similar features. PCOS has evolved in clinical understanding to encompass a heterogeneous, multifactorial, and polygenic condition most frequent endocrine disorders in reproductive-aged women [1]. Its frequency in the world is 4-21% depending on the diagnostic criteria and in India, it is 11.33% [2]. According to National Institutes of Health (NIH) 1990 criteria, it can be considered as one the most common abnormality of endocrine system in women of reproductive age.

PCOS is a heterogeneous, Multifactorial, and polygenic condition characterized by excessive androgen production by the ovaries mainly. The most common diagnostic criteria are the Rotterdam criteria, which require at least two symptoms from the following: hyperandrogenism, ovulatory dysfunction, or polycystic ovaries [3,4]. The etiology of PMOS is unknown; however, it is likely a combination of genetic and environmental factors [3]. PMOS is also associated with various metabolic abnormalities, including insulin resistance (IR), type 2 diabetes mellitus (T2DM), obesity, dyslipidemia, and an increased risk of heart disease [3,5].

Clinically, whenever PCOS is diagnosed it insinuates an increased risk for infertility, dysfunctional bleeding, endometrial carcinoma, obesity, type 2 diabetes mellitus (DM), dyslipidemia, hypertension, and possibly cardiovascular disease (CVD) Metabolic syndrome, a cluster of cardiometabolic risk factors, has been documented in approximately 65% of PCOS women presenting with clinical manifestations of hyperandrogenism as evidenced by a modified Ferriman-Gallwey score of ≥ 8 , compared to only 37.7% of those with scores below this threshold [3,7].

Material and Methods

This cross-sectional observational study was conducted in the Department of Obstetrics and Gynaecology, Jawaharlal Nehru Medical College, Aligarh Muslim University (A.M.U.), after obtaining approval from the Institutional Ethics Committee. A total of 50 women aged 18–40 years diagnosed with polycystic ovary syndrome (PCOS) according to the Rotterdam 2003 criteria were consecutively enrolled after obtaining written informed consent. Demographic details, menstrual and medical history, anthropometric measurements, and clinical features of hyperandrogenism were recorded. Laboratory investigations, including fasting blood glucose, lipid profile, liver function tests, and relevant hormonal parameters, were performed using standard laboratory methods. Pelvic ultrasonography was used to confirm polycystic ovarian morphology and classify participants into the four Rotterdam PCOS phenotypes (A, B, C, and D). Abdominal ultrasonography was performed to assess hepatic steatosis, and metabolic dysfunction-associated steatotic liver disease (MASLD) was diagnosed after excluding secondary causes of liver disease and significant alcohol consumption, in accordance with current diagnostic recommendations. The primary objective was to determine the prevalence of MASLD among women with PCOS, while the secondary objective was to evaluate its association with different PCOS phenotypes. Data were analyzed using appropriate statistical tests, with continuous variables expressed as mean $\pm$ standard deviation and categorical variables as frequencies and percentages. A p-value of <0.05 was considered statistically significant.

Results

Non-PCOS women were predominantly free of fatty liver disease (96.0%, $n=24$), while PCOS women demonstrated substantially higher rates of hepatic steatosis (32.0% with grades 1-3 combined), with 20.0% ($n=5$) exhibiting mild steatosis (Grade 1), 4.0% ($n=1$) moderate steatosis (Grade 2), and 8.0% ($n=2$) severe steatosis (Grade 3). (Figure-2)

Figure 1 shows out of 25 PCOS women, 18 women were categorised as phenotype A, 5 women were Phenotype B and Phenotype C category and only 1 woman was in phenotype D category

Table 1: Comparison of Anthropometric Parameters Between PCOS and non PCOS

Parameter	Non-PCOS Mean $\pm$ SD	PCOS Mean $\pm$ SD	P value
Age (years)	24.16 $\pm$ 5.44	22.76 $\pm$ 3.00	0.538
BMI (kg/m ²)	24.28 $\pm$ 3.12	24.55 $\pm$ 4.19	0.799
Ferriman–Gallwey score	6.24 $\pm$ 1.01	10.96 $\pm$ 2.67	<0.001
Waist circumference (cm)	86.60 $\pm$ 8.50	88.36 $\pm$ 8.80	0.402
Waist-to-hip ratio	0.86 $\pm$ 0.12	0.84 $\pm$ 0.16	0.524

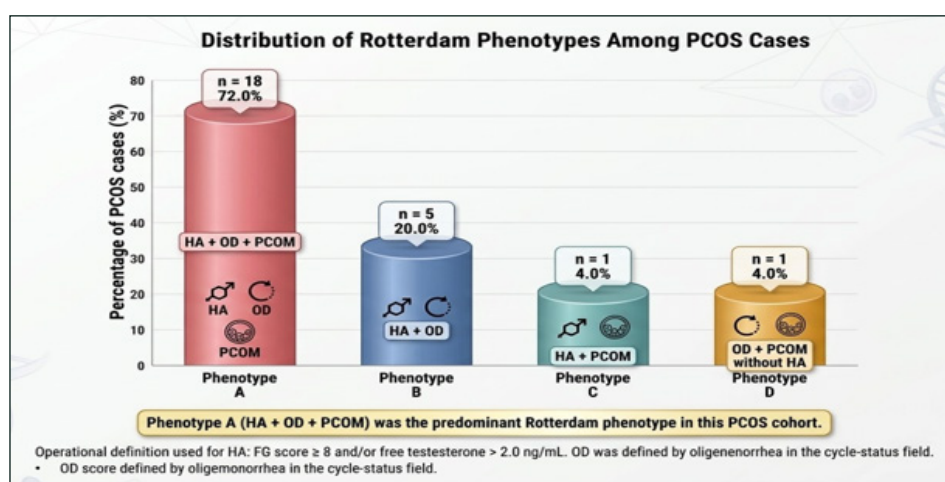


Figure 1: Phenotype Distribution Among PCOS Women

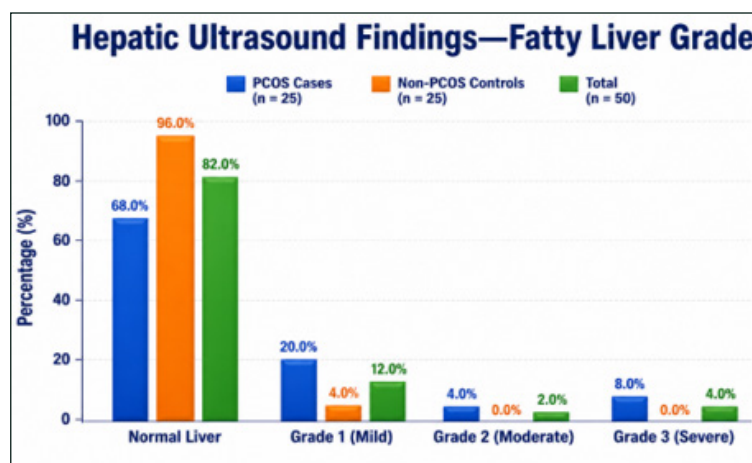


Figure 2: Grades of Fatty liver in PCOS

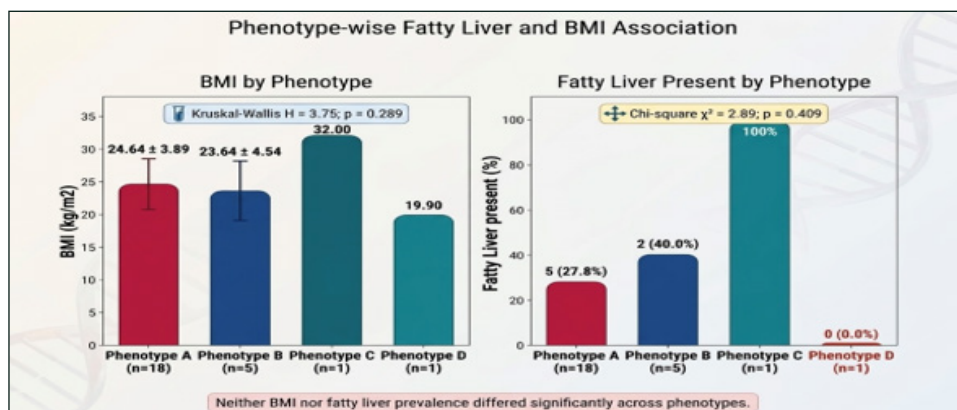


Figure 3: BMI and Fatty Liver Across Different PCOS Phenotypes

Discussion

Metabolic dysfunction are characteristic of PCOS. When dichotomized into "normal liver" versus "any fatty liver," the association achieved statistical significance ($p = 0.010$), confirming PCOS cases have significantly higher fatty liver prevalence (32.0% vs 4.0%), establishing hepatic steatosis as an important metabolic comorbidity of PCOS warranting clinical monitoring and intervention.

In terms of metabolic and hepatic comorbidity, this study observed a higher prevalence of fatty liver among PCOS women, with a significant difference when steatosis was dichotomized as present versus absent, despite similar BMI and central adiposity between groups. This pattern supports an emerging literature linking PCOS to non-alcoholic fatty liver disease and broader metabolic risk, beyond what would be expected from obesity alone [8-12]. This finding is consistent with emerging evidence linking PCOS to non-alcoholic fatty liver disease (NAFLD) and an increased metabolic risk that may extend beyond that attributable to obesity alone. In contrast, the significantly higher prevalence of any fatty liver among women with PCOS supports the hypothesis that insulin resistance and hyperandrogenism may contribute to hepatic fat accumulation independently of conventional anthropometric measures. These findings are also consistent with previous Indian studies demonstrating a substantial metabolic burden among women with PCOS and suggest that hepatic ultrasonically may have a role as an adjunctive tool for comprehensive metabolic risk assessment, particularly among women with clinical hyperandrogenism and a normal-range BMI [13,14].

Women with PCOS, as well as those with NAFLD, have an increased prevalence of metabolic syndrome and its associated risk factors, including insulin resistance, obesity, dyslipidaemia, and hypertension [15,16]. Several studies have examined the prevalence of NAFLD among women with PCOS. In one study of 85 premenopausal women with PCOS, NAFLD was identified in 55% of participants; notably, 40% of women with NAFLD had a normal BMI, highlighting that hepatic steatosis may occur in the absence of overt obesity [17]. Furthermore, a recent meta-analysis reported that women with PCOS have approximately a fourfold higher risk of developing NAFLD than healthy controls [18]. Collectively, these findings support the recognition of hepatic steatosis as an important component of the metabolic phenotype of PCOS, including among women without marked obesity.

Although increasing evidence supports an association between PCOS and NAFLD, partly due to their shared metabolic comorbidities, it remains unclear whether androgen excess independently contributes to the development of NAFLD beyond the underlying metabolic dysfunction. A large UK study involving 63,125 women evaluated risk factors for NAFLD among women with PCOS between 2000 and 2016 [19]. The study found that higher free testosterone levels and lower sex hormone-binding globulin concentrations were associated with an increased risk of NAFLD. Similarly, a cross-sectional study of 400 Chinese women with PCOS reported that a higher free androgen index, which reflects androgen bioavailability, was associated with an increased risk

of NAFLD [20]. A meta-analysis also demonstrated an association between elevated androgen levels and NAFLD in women with PCOS. Furthermore, Jones et al [21]. reported that women with PCOS and hyperandrogenism (HA) were more likely to have NAFLD than those with PCOS without HA, further supporting a potential role for androgen excess in the development of NAFLD.

It is believed that androgen excess increases IR by affecting the insulin's effect on muscle and peripheral tissues, increasing visceral adipose tissue and reducing the adiponectin, the main insulin-sensitising hormone of adipose tissue [22]. In addition, IR increases central adiposity, which increases the free fatty acids flow to the liver through the portal vein, which contributes to NAFLD formation [23].

Conclusion

The study finding suggests hyperandrogenism contributes to NAFLD formation in these women. Phenotype A was the predominant PCOS phenotype while Phenotype D was least common. Further studies are needed to assess the association of NAFLD in women with PCOS and its phenotype variants.

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