ORIGINAL ARTICLE

The Interplay of Age, Obesity Measures, and Micronutrient Deficiencies in PCOS-associated Metabolic Dysfunction Findings from a Retrospective Postobservational Cohort Study

Zubeda Tumbi[1*], Mehzamah Tumbi[2], Vaibhavi Tailor[3], Gunjan Temkar[4]

Received: 01 July 2025; *Accepted:* 01 October 2025

Abstract

Background: Polycystic ovary syndrome (PCOS) is a complex endocrine-metabolic disorder with significant age-related, anthropometric, and metabolic variations. Understanding the interplay between age, body composition, and micronutrient status can help identify predictors of metabolic dysfunction and long-term complications in women with PCOS. Most studies assess these factors in isolation, resulting in fragmented evidence and inconsistent conclusions.

Objectives: This study aimed to evaluate the predictive associations of age, anthropometric parameters, and micronutrient levels (vitamin B12 and vitamin D3) with key metabolic, cardiovascular, and nutritional markers in women diagnosed with PCOS.

Materials and methods: A cross-sectional analysis was conducted among women with PCOS, divided into two age-groups: group I (15–30 years) and group II (31–40 years). One-way analysis of variance (ANOVA) and linear regression analyses were performed to investigate age- and obesity-related differences in metabolic parameters. Pearson's correlations and regression models were applied to assess the predictive strength of age, body mass index (BMI), waist circumference (WC), vitamin B12, and vitamin D3 on metabolic outcomes. The Benjamini–Hochberg method was applied to control the false discovery rate (FDR) for multiple comparisons and ensure conclusions account for the increased risk of type I error due to multiple comparisons, thereby supporting the validity of the study's observations.

Results: The PCOS cohort exhibited generalized overweight (BMI 23.0–24.9 kg/m^2) and obesity (BMI ≥25.0 kg/m^2) in 40 and 54% of subjects, respectively; notably, visceral obesity (WC ≥80 cm) was present in 97% of the cohort, underscoring a marked predominance of central adiposity even among those with lower BMI thresholds. The PCOS cohort demonstrated high metabolic risk, with frequent insulin resistance, dyslipidemia, and hypertension; notably, 25% had nonalcoholic fatty liver disease (NAFLD), predominantly mild steatosis, indicating a substantial risk for future cardiometabolic complications. Advancing age was significantly associated with higher fasting blood sugar (FBS) ($p = 0.019$) and glycated hemoglobin (HbA1c) ($p = 0.048$), while fasting insulin declined with age ($p = 0.048$). BMI and WC were strong predictors of metabolic risk, positively impacting fasting insulin, FBS, HbA1c, and blood pressure ($p < 0.05$), while showing significant negative associations with high-density lipoprotein cholesterol (HDL-C) ($p < 0.001$). Vitamin B12 and D3 levels showed no significant impact on metabolic parameters. Vitamin B12 was predicted only by age ($p < 0.001$) and vitamin D3 ($p < 0.001$), while vitamin D3 was influenced by age ($p < 0.001$) and vitamin B12 levels ($p = 0.041$).

Conclusion: Central obesity markers—particularly WC and BMI—are robust predictors of metabolic dysfunction in PCOS, offering greater prognostic value than micronutrient levels such as vitamin B12 or D3, which showed limited association with metabolic disturbances in this population. These findings highlight the primacy of early detection and intervention targeting adiposity and insulin resistance to reduce long-term cardiometabolic risk among women with PCOS. Integrated, multifactorial risk assessment remains essential, as age, nutritional deficits, and obesity-related factors independently and collectively drive adverse metabolic outcomes in PCOS, emphasizing the need for comprehensive preventive and management strategies.

Journal of The Association of Physicians of India (2025): 10.59556/japi.73.1281

Introduction

Polycystic ovary syndrome (PCOS) is a heterogeneous endocrine disorder affecting 6–20% of women of reproductive age and is characterized by hyperandrogenism, reproductive disturbances, and metabolic dysfunction.[1] Obesity frequently coexists with PCOS, insulin resistance, and compensatory hyperinsulinemia, serving as a central driver of both its clinical manifestations and its strong association with metabolic syndrome (MetS).[2,3] The pathogenesis of MetS is closely linked to insulin resistance and chronic low-grade inflammation, which, if unaddressed, significantly increases the risk of type II diabetes and cardiovascular disease.[4] Given overlapping metabolic abnormalities, including insulin resistance, dyslipidemia, and obesity, PCOS is also now recognized as a key risk factor for nonalcoholic fatty liver disease (NAFLD), making its evaluation clinically meaningful in the present study.[5] Understanding the interplay between PCOS and MetS is essential for reducing long-term cardiometabolic risk.[5] Notably, the prevalence of MetS has risen steadily over recent decades, with its features increasingly observed as early as adolescence.[6–10] These insights underscore the urgent need for evidence-based nutritional strategies that address insulin resistance, optimize body composition, and support reproductive and metabolic health.

The combined influence of age, nutritional deficiencies, and obesity-related factors on the trajectory of PCOS remains insufficiently defined, with evidence to date being inconsistent.[11–13] This study explores these interrelationships in overweight and obese women with PCOS and identifies nutritional and metabolic predictors of disease risk and severity.

The primary objective of this study was to investigate the interrelationship between age, nutritional status, and metabolic dysfunction in overweight and obese women with PCOS. Secondary objectives included evaluating the independent effects of age, body mass index (BMI), waist circumference (WC), vitamin B12, and vitamin D3 levels on PCOS progression and severity. Each parameter was independently analyzed

[1]Founder and Director, Department of Clinical Dietetics; [2,3]Assistant Dietitian, Department of Clinical Nutrition and Dietetics; [4]Research Assistant, Department of Information and Technology, HealthWatch Nutrition Clinic, Mumbai, Maharashtra, India; *Corresponding Author

How to cite this article: Tumbi Z, Tumbi M, Tailor V, *et al.* The Interplay of Age, Obesity Measures, and Micronutrient Deficiencies in PCOS-associated Metabolic Dysfunction Findings from a Retrospective Postobservational Cohort Study. J Assoc Physicians India 2025;73(12):e8–e16.

to determine its contribution to metabolic dysfunction in PCOS. Additionally, metabolic parameters—fasting insulin levels, glycemic markers [fasting blood sugar (FBS), glycated hemoglobin (HbA1c)], lipid profile parameters [high-density lipoprotein cholesterol (HDL-C), triglycerides (TG)], and blood pressure [systolic blood pressure (SBP), diastolic blood pressure (DBP)]—were evaluated to check their interrelated impact on PCOS disease risk and severity.

Materials and Methods

This retrospective study analyzed preexisting records of 230 females aged 15–40 years who attended a nutrition clinic in Mumbai, India, between January 2020 and January 2024. Participants were recruited using purposive sampling to meet the study objectives.

Inclusion Criteria

Women aged 15–40 years diagnosed with PCOS based on the Rotterdam (2003) criteria were eligible. Diagnosis required the presence of at least two of the following: oligo- or anovulation (menstrual cycles longer than 35 days or fewer than 8 per year), clinical and/or biochemical evidence of hyperandrogenism (hirsutism, moderate-to-severe acne, or female-pattern hair loss), and polycystic ovarian morphology on ultrasound, defined as ≥12 follicles measuring 2–9 mm in diameter and/or an ovarian volume >10 cm^3 in at least one ovary.[14] Only women with complete and verifiable documentation of nutritional status and relevant metabolic parameters, as specified in the study protocol, were considered for inclusion.

Exclusion Criteria

Included pregnancy or lactation, presence of chronic systemic illness such as type I diabetes mellitus, kidney disease, cardiovascular disorders, thyroid dysfunction, or malignancy, and chronic liver disease of any origin (viral, autoimmune, alcoholic, or nonalcoholic). Women with a history of ovarian surgery or endocrine disorders resembling PCOS, including congenital adrenal hyperplasia, Cushing's syndrome, or androgen-secreting tumors, were excluded. Participants were also ineligible if they had taken medications within the past 6 months that could influence reproductive hormones, metabolism, or liver function (e.g., hormonal contraceptives, antiandrogens, insulin sensitizers, lipid-lowering drugs, or corticosteroids). Current use of vitamin supplementation or ongoing insulin therapy was also an exclusion criterion.

Anthropometric Measurements

Standardized procedures were used to assess and record anthropometric measurements to ensure both accuracy and reduced variability during the data collection process. Anthropometric data consisted of height, weight, BMI, and WC.

The height of participants was measured in centimeters using a stadiometer installed in the clinic using the following protocol—barefoot standing position while maintaining an erect posture and aligning the head with the Frankfurt horizontal plane.[15] Body weight was measured using a calibrated Weightron weighing scale (Model No. IND/09/05/397), with readings rounded to the nearest 100 gm.[15] Participants were weighed wearing light clothing and without shoes to ensure accuracy.[15] BMI was calculated using the standard formula: BMI (kg/m^2) = weight (kg) / height^2 (m^2). BMI serves as a simple, cost-effective anthropometric indicator for estimating body fat and is widely used to classify individuals as overweight or obese based on established cutoff values. WC was measured using a nonstretchable, flexible measuring tape to ensure accuracy. WC was measured midway between the last palpable rib and the iliac crest after normal expiration to ensure consistency and reduce measurement error. Measurements were recorded to the nearest 0.1 cm to enhance precision and minimize variability.[16]

Blood Pressure Measurements

Blood pressure readings were recorded using an Omron automatic blood pressure monitor, model HEM-7156. Two BP readings were recorded from the participant in the supine position after resting comfortably for at least 5–10 minutes, with the average value determined as the final BP measurement. Participants were classified as hypertensive if they exhibited a SBP of 130 mm Hg or above, a DBP of 85 mm Hg or above, or were undergoing treatment with antihypertensive medications.

Biochemical and Laboratory Assessments

Biochemical parameters were extracted from patients' clinical records maintained at the Nutrition Clinic: (1) metabolic markers—fasting insulin, FBS, HbA1c, TG, and HDL-C, and (2) serum vitamin B12 and vitamin D3 test records to evaluate micronutrient status. The micronutrient cutoff values commonly used for diagnosing deficiency, for serum vitamin B12 <200 pg/mL and serum 25(OH) D <30 ng/mL, are widely accepted standards, and research supports their association with increased metabolic risk factors.[17,18]

Ultrasound Evaluation of the Abdomen

Ultrasound findings were retrieved from patients' medical records and comprised evaluations of hepatic steatosis and ovarian morphology. Fatty liver was classified from grades 1 to 3 using abdominal ultrasound criteria, which included increased hepatic echogenicity, decreased visualization of intrahepatic vessels, and blurring of the diaphragm—findings that indicate hepatic steatosis. Ovarian morphology, including follicular characteristics, was recorded from pelvic ultrasound reports conducted as part of the diagnostic assessment at the start of nutrition counseling.[19]

The study utilized anonymized retrospective data, with informed consent obtained at the entry point in accordance with national guidelines. The study was qualified for exemption from formal ethics review, and all procedures conformed to the Declaration of Helsinki.

Statistical Analysis

Anonymized data was compiled using Microsoft Excel 2016. Descriptive statistics were calculated in Excel, and inferential analyses were performed using Jamovi (v2.6.23). A one-way analysis of variance (ANOVA) was used to compare PCOS-related clinical variables between women aged 15–30 and 31–40 years. Pearson's correlation assessed associations among anthropometric, metabolic, and nutritional parameters. Linear regression evaluated the effects of age, BMI, WC, vitamin B12, and vitamin D3 on metabolic health markers. Multiple hypothesis testing was corrected using the false discovery rate (FDR) control approach (Benjamini–Hochberg procedure), implemented in Python 3 (statsmodels package) within the Visual Studio Code IDE. Corrected q-values were compared against a significance threshold of 0.05.[20]

Results

Baseline Characteristics of the Study Population

The study included 230 women aged 15–40 years, all diagnosed with PCOS (Table 1). Participants were stratified into two age-groups: group I (15–30 years) and group II (31–40 years). The overall mean age was 28.3 ± 6.3 years. The mean body weight was 81.5 ± 15.7 kg, with a corresponding mean BMI of 31.2 ± 5.6 kg/m^2, classifying the population within the overweight to obese range. Based on BMI categories, 6% of participants were of normal weight, 40% were overweight, and 54% were obese. The

mean WC was 100.1 ± 11.3 cm, with only 3% of participants having a WC <80 cm, while the majority (97%) exceeded the ≥80 cm threshold, indicating a high prevalence of central obesity.

The Polycystic Ovary Study Cohort Exhibited Significant Metabolic Abnormalities

About 31% insulin resistance, 7% type 2 diabetes mellitus (T2DM), 13% hypertension, 31% elevated TG, and 76% low HDL-C levels. NAFLD was identified in 25% of participants, comprising 17% with grade 1, 6% with grade 2, and 2% with grade 3 steatosis.

Significant nutritional deficiencies were observed in this PCOS cohort, with 23% (n = 52) having low serum vitamin B12 levels and 69% (n = 159) with deficient vitamin D3 status.

The study cohort exhibited the following signs and symptoms of hyperandrogenism—hirsutism 30%, acne 41%, hair thinning 62%, and acanthosis nigricans 43%.

The study participants with PCOS were divided into two age-groups: group I (15–30 years) and group II (31–40 years). Classification was based on standard clinical thresholds for BMI and WC. Figure 1 shows BMI distribution across groups—in group I, 6% had normal BMI, 39% were overweight, and 55% were obese; in group II, 4% had normal BMI, 43% were overweight, and 53% were obese. Based on the WHO Asian-specific WC cutoff (≥80 cm), 96% of participants in group I and 100% in group II had abdominal obesity (Fig. 2). Based on the WHO Asian-specific cutoff for WC (≥80 cm), visceral obesity was present in 96% of women aged 15–30 years and in 100% of women aged 31–40 years (Fig. 2).

Table 1: Baseline characteristics of PCOS women in the study sample

Baseline parameters	*Value (n = 230)*
Age (years) (mean ± SD)	28.3 ± 6.3
Weight (kg) (mean ± SD)	81.5 ± 15.7
BMI (kg/m^2) (mean ± SD)	31.2 ± 5.6
WC (cm) (mean ± SD)	100.1 ± 11.3
Individuals with insulin resistance	71 (31%)
Individuals with T2 diabetes	18 (7%)
Individuals with hypertension	30 (13%)
Individuals with elevated TG	71 (31%)
Individuals with low HDL-C	174 (76%)
Individuals with low vitamin B12	52 (23%)
Individuals with low vitamin D3	159 (69%)
Individuals with NAFLD	56 (25%)
Individuals with acanthosis nigricans	99 (43%)
Individuals with hirsutism	68 (30%)
Individuals with acne	94 (41%)
Individuals with hair thinning	143 (62%)

N (%) = Number (percentage)

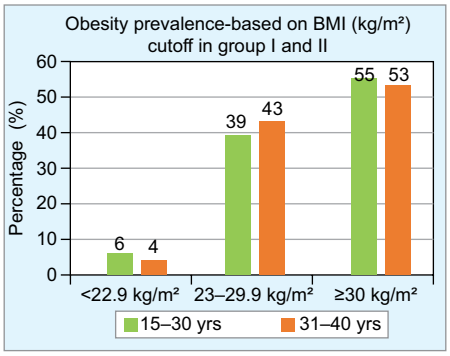

Fig. 1: Obesity prevalence-based on BMI (kg/m^2) cutoff in groups I and II

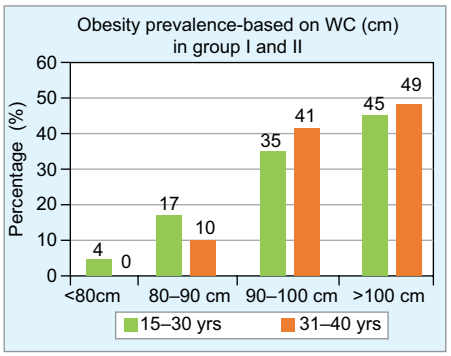

Fig. 2: Obesity prevalence-based on WC (cm) in groups I and II

Age-stratified Analysis of Metabolic and Biochemical Parameters

Anthropometric Parameters

A one-way ANOVA was conducted to compare anthropometric parameters between group I (15–30 years) and group II (31–40 years) (Table 2). An FDR correction was applied to account for multiple comparisons. BMI was 31.43 ± 5.61 kg/m^2 in group I and 30.99 ± 5.57 kg/m^2 in group II (p = 0.568; FDR q = 0.050). WC was 99.49 ± 11.59 cm in group I and 101.3 ± 10.96 cm in group II (p = 0.238; FDR q = 0.036).

Metabolic Components

Mean values of anthropometric, cardiometabolic, and micronutrient status of PCOS components of the cohort are shown in Table 2. Comparative analyses identified statistically significant differences in certain variables; however, significance was determined based on FDR-adjusted q-values to appropriately control multiple hypothesis testing. Reporting both unadjusted and adjusted p-values enhances transparency and ensures that conclusions account for the increased risk of type I

Table 2: Analysis of age-dependent variations in anthropometric, cardiometabolic, and micronutrient status in PCOS

Independent variables	*15–30 years (n = 139)*	*31–40 years (n = 91)*	*p-value*	*FDR-adjusted q-value*
BMI, kg/m^2	31.43 ± 5.61	30.99 ± 5.57	0.568	0.050
WC, cm	99.49 ± 11.59	101.3 ± 10.96	0.238	0.036
SBP, mm Hg	113.89 ± 11.35	117.89 ± 12.48	0.013	0.014
DBP, mm Hg	76.26 ± 8.45	78.65 ± 10.41	0.057	0.023
HbA1c, %	5.52 ± 0.6	5.7 ± 0.79	0.046	0.018
FBS, mg/dL	90.84 ± 16.9	93.54 ± 19.69	0.268	0.041
Fasting insulin, µIU/mL	20.82 ± 14.94	17.51 ± 10.75	0.07	0.027
Triglyceride, mg/dL	131.35 ± 74.95	145.49 ± 70.26	0.154	0.032
HDL cholesterol, mg/dL	43.3 ± 9.7	44.48 ± 11.45	0.4	0.045
Vitamin B12, pg/mL	279.36 ± 148.46	343.09 ± 153.13	0.002	0.005
Vitamin D3, ng/mL	21.06 ± 17.55	28.55 ± 20.25	0.003	0.009

Comparative analyses demonstrated significant differences in variables; significance determined using FDR-adjusted q-values to control for multiple testing, and raw p-values are reported for transparency

error due to multiple comparisons, thereby supporting the validity of the study's observations.

Body mass index was measured in kg/m^2 to assess overall adiposity, with no significant difference between the 15–30 and 31–40-year groups (p = 0.568, FDR q = 0.050), indicating stability across these cohorts. WC was recorded in centimeters as an indicator of central obesity, which did not differ significantly across age-groups (p = 0.238, FDR q = 0.036). Significant age-dependent differences were observed in SBP (raw p = 0.013; FDR q = 0.014) and vitamin B12 levels (raw p = 0.002; FDR q = 0.005) among participants with PCOS. SBP and DBP were measured in mm Hg; systolic pressure showed a significant increase with age (p = 0.013, q = 0.014), while diastolic pressure approached significance (p = 0.057, q = 0.023). HbA1c (%) as a marker of long-term glycemic control was higher in the older age-group with statistically significant differences post-FDR adjustment (p = 0.046, q = 0.018). FBS levels (mg/dL) showed no significant variation between age-groups (p = 0.268, q = 0.041). Fasting insulin (μIU/mL), an insulin resistance indicator, trended lower in the older cohort but did not reach significance after correction (p = 0.07, q = 0.027). Lipid profile components included TG (mg/dL), which were higher in older women but not statistically significant after FDR correction (p = 0.154, q = 0.032), and HDL cholesterol (mg/dL), which showed no age-related difference (p = 0.4, q = 0.045). The incidence of fatty liver increased with age, with grade 1 steatosis observed in 14 and 20% of group I and group II, while 7% in both groups exhibited fatty liver grades 2–3.

Micronutrient Parameters

Vitamin B12 (pg/mL) and vitamin D3 (ng/mL) levels, important micronutrients implicated in PCOS pathophysiology, were assessed. Vitamin B12 mean values were significantly higher in the 31–40 years group (raw p = 0.002; FDR-adjusted q = 0.005), and vitamin D3 means also showed a similar statistically significant increase in the older age-group (raw p = 0.003; FDR-adjusted q = 0.009) after correction for multiple testing.

Correlative Trends between Age and Metabolic Risk Variables in Women with PCOS

Pearson's correlation analysis between age and metabolic, biochemical, and anthropometric parameters is presented in Table 3. FBS correlated positively with age (r = 0.155, p = 0.019), as did HbA1c (r = 0.131, p = 0.048). Fasting insulin correlated negatively with age (r = −0.176, p = 0.008). Vitamin B12 (r = 0.226, p < 0.001) and vitamin D3 (r = 0.231, p < 0.001) also showed significant positive correlations with age. No significant correlations were observed for WC, BMI, SBP, DBP, TG, or HDL-C (Table 3).

Predictive Analysis of Age, Anthropometric, and Micronutrient Parameters in PCOS

This study examined how age, body measurements (BMI, WC), and micronutrient status (serum vitamin B12 and D3) relate to metabolic and clinical features in women with PCOS. Using linear regression analysis, several significant associations emerged, underscoring the independent and combined influence of these variables on cardiometabolic and nutritional risk markers.

Predictive Analysis of Age on Metabolic and Nutritional Status in PCOS

Linear regression analysis of the study cohort demonstrated that age was a significant predictor of glycemic indices—FBS (β = 0.155, 95% CI: 0.03, 0.28, p = 0.019) and HbA1c (β = 0.131, 95% CI: 0.00, 0.26, p = 0.048). Age was inversely associated with fasting insulin (β = −0.176, 95% CI: −0.30, −0.05, p = 0.008). Age was a significant predictor of serum vitamin B12 (β = 0.226, 95% CI: 0.10, 0.35, p < 0.001) and vitamin D3 levels (β = 0.231, 95% CI: 0.10, 0.36, p < 0.001), indicating a positive association between increasing age and higher concentrations of these vitamins. Age was not significantly associated with BMI, WC, SBP or DBP, TG, or HDL cholesterol levels (Fig. 3).

Predictive Analysis of BMI on Metabolic Parameters

The linear regression model identified BMI as a significant predictor of various metabolic, anthropometric, and biochemical parameters within the PCOS cohort studied. Specifically, BMI strongly predicted WC (β = 0.772, 95% CI: 0.69, 0.86, p < 0.001). Higher BMI was also significantly associated with increased fasting insulin (β = 0.319, 95% CI: 0.20, 0.44, p < 0.001), fasting blood glucose (β = 0.219, 95% CI: 0.09, 0.35, p < 0.001), and HbA1c (β = 0.215, 95% CI: 0.09, 0.34, p < 0.001). Additionally, BMI significantly predicted systolic (β = 0.223, 95% CI: 0.10, 0.35, p < 0.001) and DBP (β = 0.303, 95% CI: 0.18, 0.43, p < 0.001). Conversely, BMI exhibited a negative association with HDL cholesterol (β = −0.212, 95% CI: −0.34, −0.08, p = 0.001). No significant association was found between BMI and age (β = −0.076, 95% CI: −0.21, 0.05, p = 0.251) (Fig. 4).

Predictive Analysis of Waist Circumference on Metabolic Parameters

In the PCOS cohort, linear regression analysis showed that WC significantly predicted BMI

Table 3: Pearson's correlation heatmap comparing metabolic health parameters in women with PCOS

	Age	*BMI*	*WC*	*SBP*	*DBP*	*FBS*	*HbA1c*	*F insulin*	*Vit B12*	*Vit D3*	*TG*	*HDL-C*
Age	–											
BMI	−0.076	–										
WC	0.056	0.772***	–									1
SBP	0.109	0.223***	0.216***	–								
DBP	0.122	0.303***	0.286***	0.691***	–							
FBS	0.155*	0.219***	0.212**	0.076	0.159*	–						
HbA1c	0.131*	0.215**	0.182**	0.072	0.176**	0.701***	–					
F insulin	−0.176**	0.319***	0.297***	0.130*	0.121	0.254***	0.211**	–				−1
Vit B12	0.226***	−0.002	−0.03	−0.06	−0.001	0.056	0.02	0.031	–			
Vit D3	0.231***	−0.048	−0.033	−0.011	0	0.068	0.001	−0.034	0.319***	–		
TG	0.099	0.052	0.099	0.232***	0.291***	0.151*	0.11	0.183**	0.105	0.045	–	
HDL-C	0.097	−0.212**	−0.159*	−0.124	−0.167*	−0.236***	−0.249***	−0.218***	0.055	−0.011	−0.342***	–

*Significant at p < 0.05, **Significant at p < 0.01, ***Significant at p < 0.001; BMI, body mass index; DBP, diastolic blood pressure; F insulin, fasting insulin; FBS, fasting blood sugar; HbA1c, glycated hemoglobin; HDL-C, high density lipoprotein-cholesterol; SBP, systolic blood pressure; TG, triglycerides; Vit B12, vitamin B12; Vit D3, Vitamin D3; WC, waist circumference

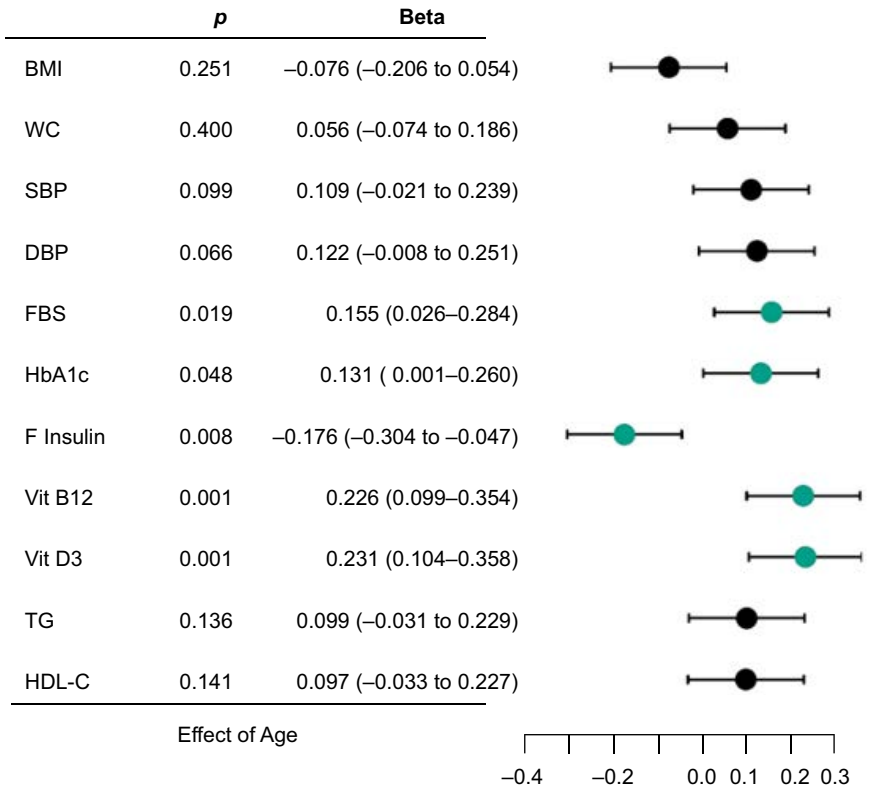

Fig. 3: Linear regression analysis depicting the effect of age on anthropometric, metabolic, and nutritional parameters in women with PCOS

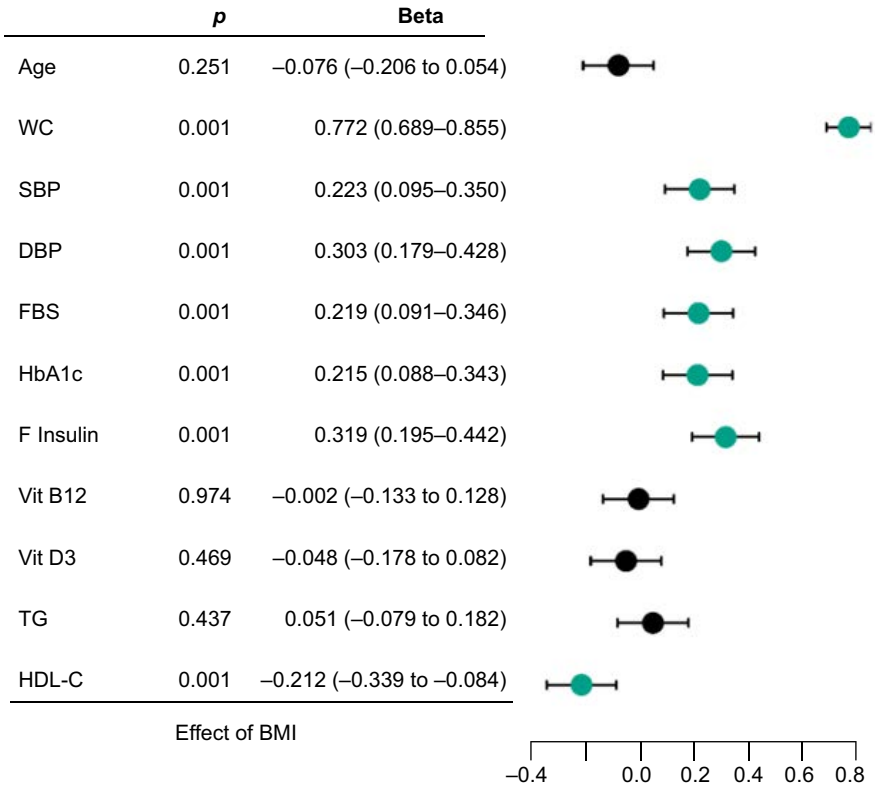

Fig. 4: Linear regression analysis depicting the effect of BMI on anthropometric, metabolic, and nutritional parameters in women with PCOS

($\beta = 0.772$, 95% CI: 0.69, 0.86, $p < 0.001$). WC also predicted a significant association with SBP ($\beta = 0.216$, 95% CI: 0.09, 0.34, $p < 0.001$) and DBP ($\beta = 0.286$, 95% CI: 0.16, 0.41, $p < 0.001$).

Glycemic indices were associated with WC, including FBS ($\beta = 0.212$, 95% CI: 0.08, 0.34, $p = 0.001$), HbA1c ($\beta = 0.182$, 95% CI: 0.05, 0.31, $p = 0.006$), and fasting insulin ($\beta = 0.297$, 95% CI: 0.17, 0.42, $p < 0.001$). WC negatively predicted HDL-C ($\beta = -0.159$, 95% CI: −0.29, −0.03, $p = 0.016$). No significant associations were observed between WC and age ($\beta = 0.0558$, 95% CI: −0.07, 0.19, $p = 0.400$), vitamin B12 ($\beta = -0.0297$, 95% CI: −0.16, 0.10, $p = 0.654$), vitamin D3 ($\beta = -0.0334$, 95% CI: −0.16, 0.10, $p = 0.614$), or TG ($\beta = 0.0988$, 95% CI: −0.03, 0.23, $p = 0.135$) (Fig. 5).

Predictive Analysis of Vitamin B12 on Nutritional and Metabolic Parameters

In the PCOS cohort, linear regression analysis showed that vitamin B12 was a significant predictor of vitamin D3 levels ($\beta = 0.319$, 95% CI: 0.20, 0.44, $p < 0.001$) and age ($\beta = 0.226$, 95% CI: 0.10, 0.35, $p < 0.001$). No significant associations were observed between vitamin B12 and WC ($\beta = -0.0297$, 95% CI: −0.16, 0.10, $p = 0.654$) or BMI ($\beta = -0.00214$, 95% CI: −0.13, 0.13, $p = 0.974$). Vitamin B12 did not significantly predict SBP ($\beta = -0.0598$, 95% CI: −0.19, 0.07, $p = 0.367$) or DBP ($\beta = -0.0012$, 95% CI: −0.13, 0.13, $p = 0.986$). No significant associations were found with fasting insulin ($\beta = 0.0314$, 95% CI: −0.10, 0.16, $p = 0.636$), FBS ($\beta = 0.0564$, 95% CI: −0.07, 0.19, $p = 0.394$), or HbA1c ($\beta = 0.0197$, 95% CI: −0.11, 0.15, $p = 0.767$). Vitamin B12 did not significantly predict TG ($\beta = 0.105$, 95% CI: −0.02, 0.24, $p = 0.113$) or HDL-C ($\beta = 0.0548$, 95% CI: −0.08, 0.19, $p = 0.408$) (Fig. 6).

Predictive Analysis of Vitamin D3 on Nutritional and Metabolic Parameters

Linear regression analysis identified vitamin D3 levels as a significant predictor of age ($\beta = 0.231$, 95% CI: 0.10, 0.36, $p < 0.001$) and vitamin B12 ($\beta = 0.319$, 95% CI: 0.20, 0.44, $p < 0.001$). No significant associations were observed between vitamin D3 and BMI ($\beta = -0.048$, 95% CI: −0.18, 0.08, $p = 0.469$) or WC ($\beta = -0.0334$, 95% CI: −0.16, 0.10, $p = 0.614$). Vitamin D3 did not significantly predict FBS ($\beta = 0.0675$, 95% CI: −0.06, 0.20, $p = 0.308$, HbA1c ($\beta = 0.00106$, 95% CI: −0.13, 0.13, $p = 0.987$), or fasting insulin ($\beta = -0.0337$, 95% CI: −0.16, 0.10, $p = 0.612$). No significant predictive relationships were found between vitamin D3 and TG ($\beta = 0.045$, 95% CI: −0.09, 0.18, $p = 0.497$) or HDL-C ($\beta = -0.0107$, 95% CI: −0.14, 0.12, $p = 0.872$) (Fig. 7).

Discussion

Women diagnosed with PCOS have an increasing prevalence of multimorbidity over time, which is associated with a range of long-term health risks that may affect quality of life.[21] Our study group demonstrated a

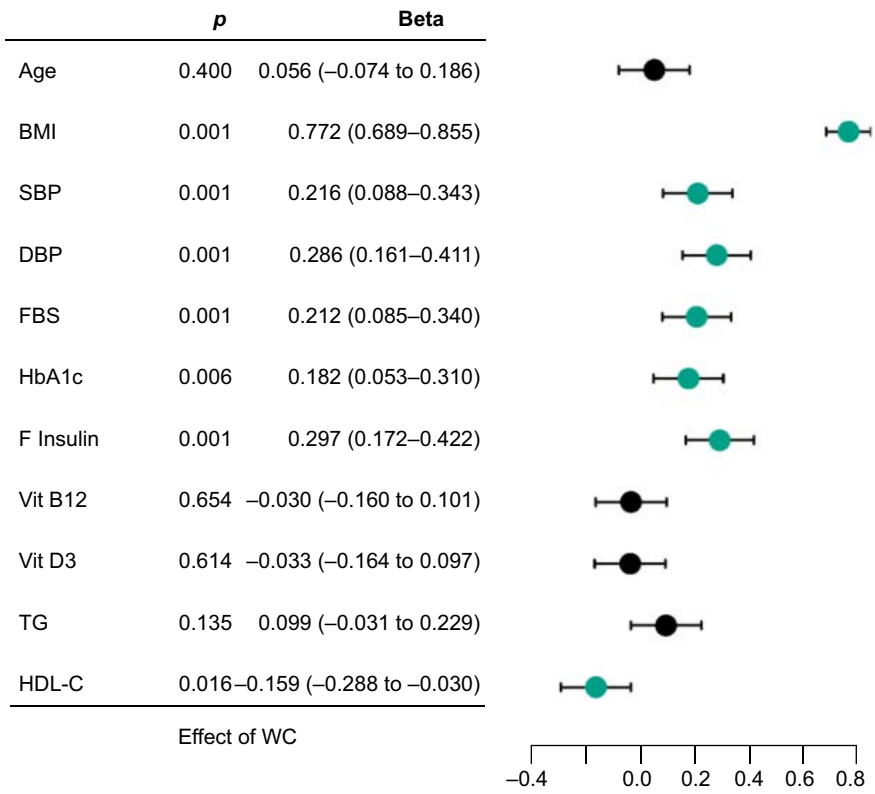

Fig. 5: Linear regression analysis depicting the effect of WC on anthropometric, metabolic, and nutritional parameters in women with PCOS

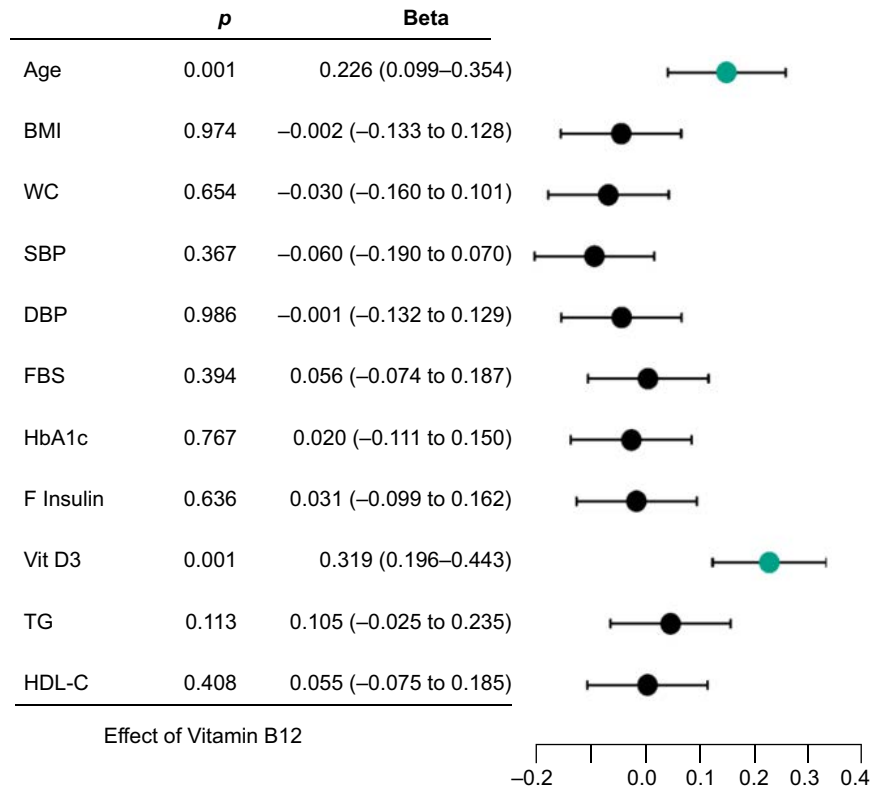

Fig. 6: Linear regression analysis depicting the effect of vitamin B12 on anthropometric, metabolic, and nutritional parameters in women with PCOS

clear age-related worsening of metabolic and cardiovascular parameters, highlighting the progressive nature of the condition. This conclusion is further supported by the use of FDR correction, which minimizes false-positive results and thereby strengthens the validity of our findings. Advancing age was markedly associated with higher FBS levels ($p = 0.019$) and HbA1c levels ($p = 0.048$), indicating impaired glycemic control. The prevalence of hypertension was 7% in the older group and 6% in the younger group, indicating an increase with age. These results underscore the heightened metabolic risk observed in older women with PCOS. Consistent with our findings, Agarwal et al. observed an increase in the prevalence of diabetes from 24% in women under 40 years to 28% in those over 40 years, while the prevalence of hypertension rose from 13 to 28%. Age-related decline was also observed in impaired oral glucose tolerance (from 48 to 57%). Older women with PCOS should be afforded the opportunity to age in good health rather than being constrained by cumulative disease burden, underscoring the critical need for further investigation.[22]

Age emerged as a significant predictor of glycemic indices in our cohort, with FBS increasing with age ($p = 0.019$), alongside HbA1c ($p = 0.048$). These findings suggest a progressive decline in glucose metabolism with advancing age among women with PCOS. Supporting this, Gautam et al. reported significantly higher FBS (98.8 ± 21.4 vs 90.1 ± 13.7 mg/dL, $p = 0.001$) and HbA1c levels (5.6 ± 0.8 vs 4.8 ± 0.4%, $p < 0.001$) in the PCOS group compared to controls. Their study also observed positive associations of FBS ($\beta = 0.155$, $p = 0.019$) and HbA1c ($\beta = 0.131$, $p = 0.048$) with age, consistent with our results. Together, these findings reinforce the notion that age-related beta-cell dysfunction and reduced insulin sensitivity may contribute to worsening glycemic control over time in women with PCOS.[23]

In our study, fasting insulin levels were negatively correlated with age ($r = -0.176$, $p = 0.008$), suggesting a decline in compensatory hyperinsulinemia among older women with PCOS. Elevated fasting insulin levels (≥20 µIU/mL) were more prevalent in group I (35%) than in group II (25%). Mean TG levels were modestly higher in group II compared to group I ($p = 0.154$, FDR $q = 0.032$), with a higher prevalence of elevated TG (≥150 mg/dL) in older participants (37 vs 27%), indicating an age-related impact on lipid imbalance. Group II also showed higher mean HbA1c levels ($p = 0.046$, FDR $q = 0.018$), with the prevalence of elevated HbA1c (≥5.7%) rising from 28% in younger women to 40% in older women.

These age-related patterns reflect a disease progression timeline in PCOS—beginning with early compensatory hyperinsulinemia and advancing toward

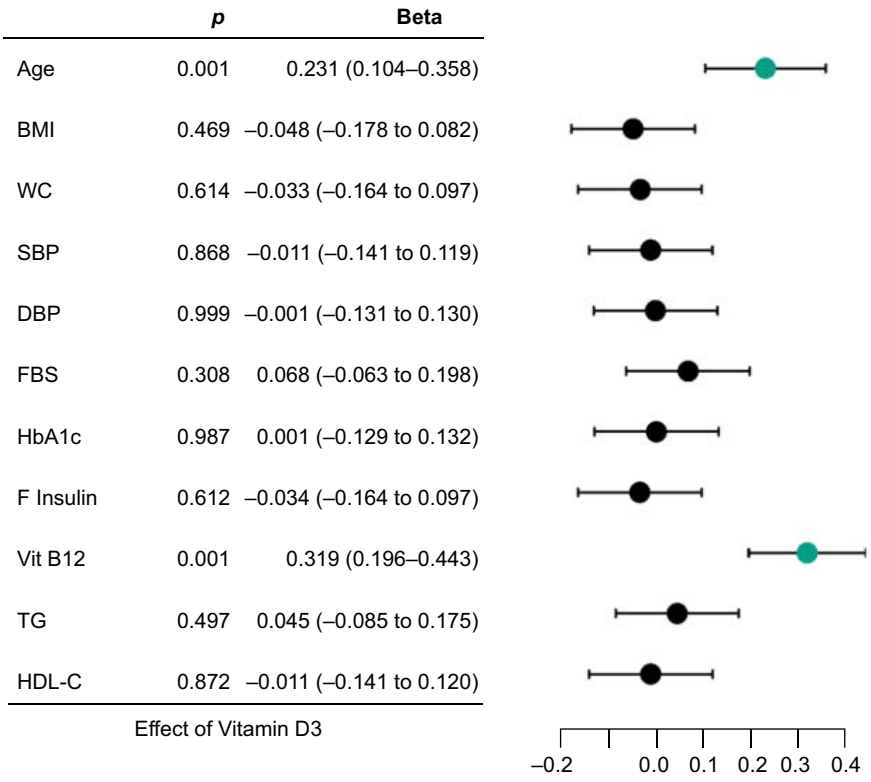

Fig. 7: Linear regression analysis depicting the effect of vitamin D3 on anthropometric, metabolic, and nutritional parameters in women with PCOS

overt glycemic and lipid abnormalities. This trajectory is consistent with findings from Amisi, who reported that insulin levels are initially elevated to counteract insulin resistance, but beta-cell dysfunction over time contributes to worsening glucose and lipid profiles. Mansour et al. further supported this pattern, showing a strong correlation between HOMA-IR and fasting insulin (r = 0.975, $p < 0.001$) in younger women, while older women exhibited higher fasting glucose and dyslipidemia. Together, these findings suggest that metabolic deterioration in PCOS follows a progressive course, and our cross-sectional data reflects this continuum as described in longitudinal studies.[24,25]

In our regression model, BMI was strongly predicted by WC ($\beta = 0.772$, 95% CI: 0.69, 0.86, $p < 0.001$), underscoring the close relationship between overall and central adiposity in women with PCOS. This strong predictive association highlights the central role of abdominal fat accumulation in contributing to increased BMI and related metabolic disturbances. Supporting our findings, Singh et al. reported a strong positive correlation between WC and BMI ($r = 0.75$) in their cohort, with a mean WC of 68.69 cm and a mean BMI of 23.07 kg/m^2. The consistent relationship between WC and BMI across studies reinforces the importance of central adiposity as a key determinant of metabolic risk in PCOS. Given that visceral fat is closely linked to insulin resistance, dyslipidemia, and cardiovascular complications, WC serves as a critical and accessible marker for assessing metabolic health in this population.[26]

In our study, BMI was a significant predictor of several metabolic parameters in women with PCOS. Higher BMI was associated with increased FBS ($p < 0.001$), HbA1c (p = 0.001), SBP and DBP (both $p < 0.001$), and lower HDL-C ($p = 0.001$).

Waist circumference also inversely predicted HDL-C ($\beta = -0.159$, 95% CI: −0.29, −0.03, $p = 0.016$), reflecting the decline in HDL-C with increasing central adiposity and metabolic risk. These findings are supported by Soares et al., who observed higher BMI in women with MetS (28.19 ± 4.30 vs 24.61 ± 4.31, $p < 0.001$), along with Sharma et al., who reported a mean HDL-C of 52.16 ± 6.32 mg/dL and WC of 87.35 ± 11.50 cm, with a significant inverse correlation between HDL-C and WC ($p < 0.05$). Likewise, Sun et al. demonstrated the diagnostic utility of WC and HDL-C for MetS, with an AUC of 0.882, highlighting their value in metabolic risk assessment.[27–29]

Waist circumference emerged as a significant predictor of metabolic abnormalities in this study. The older female cohort exhibited a higher mean WC (101.3 ± 10.96 cm) compared to the younger cohort (99.49 ± 11.59 cm), both indicating central obesity. Linear regression analysis demonstrated a positive association between WC and SBP ($p < 0.001$) and DBP ($p < 0.001$) blood pressure, FBS ($p = 0.001$), HbA1c (p = 0.006), and fasting insulin ($p < 0.001$). Conversely, a negative association was observed between WC and HDL cholesterol ($p = 0.016$). These results suggest the role of central obesity in contributing to cardiometabolic risk in PCOS, consistent with findings from a study by Pazderska et al., which indicated that WC is a more reliable surrogate marker for cardiometabolic risk than PCOS status alone. The study found that many cardiometabolic abnormalities in women with PCOS were primarily associated with central obesity. After adjusting for WC, these abnormalities did not show significant differences compared to women without PCOS. This underscores the importance of WC as a predictor of cardiometabolic risk in reproductive-age women, potentially being more relevant than the diagnostic significance of PCOS status itself.[30]

In our study, the most frequent marker of metabolic dysfunction among women with PCOS was reduced HDL-C (<50 mg/dL), observed in 174 (76%) of participants. This was followed by central obesity (WC >80 cm) in 224 (97%), elevated TG (TG ≥150 mg/dL) in 71 (31%), elevated FBS (≥100 mg/dL) in 39 (18%), and hypertension in 30 (13%). These findings are consistent with those reported by Giri et al., who identified low HDL-C (<50 mg/dL) as the most prevalent component of MetS in women with PCOS, observed in 90 participants (84.9%). This was followed by central obesity in 60 (56.6%), hypertriglyceridemia in 47 (44.33%), elevated FBS in 34 (32.07%), and hypertension in 14 participants (13.2%).[31]

In our cohort, BMI had a significant positive association with FBS ($p < 0.001$) and HbA1c ($p = 0.001$), and a significant negative association with HDL-C ($p = 0.001$). This indicates the metabolic influence of increasing adiposity on glycemic control and lipid metabolism in women with PCOS. Gudiseva et al. identified statistically significant positive correlations between BMI and FBS, HbA1c, and lipid profile at a 5% p-value. Statistically significant negative correlations were observed between HDL and BMI, FBS, HbA1c, and TG at a 5% p-value. Women with PCOS have a higher likelihood of dyslipidemia, which is a determinant of cardiovascular diseases; therefore, reducing dyslipidemia plays a role in the primary prevention of CVD. Consequently, they should be routinely screened for lipid profiles and hyperglycemic states to prevent metabolic disorders.[32]

Our study identified strong, statistically significant positive correlations between WC and key glycemic indicators in women with PCOS. Specifically, each centimeter increase in WC was associated with higher FBS levels ($p = 0.001$) and HbA1c ($p = 0.006$) and demonstrated an even stronger association with elevated fasting insulin levels ($p < 0.001$). Our findings highlight visceral adiposity as a key determinant of insulin resistance and are consistent with the findings of Saravia et al., who reported that among females with MetS ($N = 735$), elevated HbA1c was primarily associated with hyperglycemia and insulin resistance. Elevated insulin levels showed a significant positive correlation with WC.[33]

In this cohort, 25% of participants had NAFLD, with 17% classified as grade 1, 6% as grade 2, and 2% as grade 3. The overall prevalence of NAFLD among women with PCOS was 43%, indicating a high burden of hepatic involvement despite the relatively young age of participants. This suggests that both metabolic dysfunction and PCOS-specific factors contribute to NAFLD occurrence. Implementation of screening programs could support the early identification of metabolic-associated fatty liver disease and mitigate its consequences. Additional studies are needed to clarify the burden of liver-related outcomes as NAFLD advances in women with PCOS. This is consistent with Manzano-Nunez et al.'s finding of a 43% prevalence of NAFLD in women with PCOS having insulin resistance and obesity, highlighting a high risk in this population. Both studies emphasize the need for early screening, particularly in high-risk groups like those with PCOS, to detect and manage NAFLD before it progresses to more severe stages. Future studies are required to clarify the trajectory of long-term liver outcomes and to evaluate whether targeted screening programs can effectively reduce associated morbidity.[5]

In our cohort, 23% of women exhibited vitamin B12 insufficiency and 69% were deficient in vitamin D3, reflecting a considerable burden of micronutrient deficiencies. However, despite their high prevalence, neither vitamin significantly predicted central obesity (BMI, WC), blood pressure, glycemic markers (fasting insulin, FBS, HbA1c), or lipid parameters (TG, HDL-C). These findings suggest that although micronutrient deficiencies are frequent in women with PCOS, they do not act as independent determinants of metabolic dysfunction. Instead, metabolic risk appears to be more strongly driven by adiposity and insulin resistance rather than vitamin status alone.

Our findings align with Carrasco-Cabezas et al., who found no correlation between low B12 and BMI or insulin resistance, despite more than 60% deficiency rates. Similarly, Ulloque-Badaracco et al. reported no significant B12 difference between PCOS and controls (SMD = −0.11, $p = 0.13$) across 17 studies. Su et al., using Mendelian randomization, found no causal link between genetically predicted B12 levels and PCOS or its metabolic traits.[34–36] Vitamin D3 showed no significant associations with metabolic or anthropometric measures in our cohort of PCOS women. Age and vitamin B12 were the only independent predictors of vitamin D3 status, underscoring the influence of both micronutrient interplay and age-related physiology on vitamin D3 levels. This is consistent with Białka-Kosiec et al. and Kim JJ et al., who found no significant associations between vitamin D and clinical, metabolic, or hormonal features of PCOS.[37,38] Together, these results indicate that while vitamin B12 and D3 deficiencies are common in PCOS, they do not appear to influence its metabolic phenotype. Screening remains important, but supplementation should be individualized rather than assumed to modify metabolic risk.

In our regression model, a significant bidirectional relationship was observed between vitamin B12 and vitamin D3 levels—each independently predicted the other. This finding is consistent with observations by Shen et al., who reported a positive correlation between the two vitamins in PCOS. They suggested this association may be attributed to shared deficiency pathways, such as gut microbiota dysbiosis and malabsorption syndromes prevalent in PCOS, or to cosupplementation practices, rather than a direct metabolic interaction. These insights highlight the importance of considering underlying nutritional and gastrointestinal factors when evaluating micronutrient status in PCOS.[39]

In our cohort, clinical signs of hyperandrogenism and insulin resistance were common—hirsutism (30%), acne (41%), hair thinning (62%), and acanthosis nigricans (43%). These features reflect the hormonal and metabolic disturbances characteristic of PCOS. Comparable findings were reported by Soares et al., who observed hirsutism in 28.3%, acne in 9.2%, and acanthosis nigricans in 15.8% of their cohort. Notably, the prevalence of hirsutism (42.22 vs 20%) and acanthosis nigricans (28.8 vs 8%) was significantly higher in those with MetS. The overlap between clinical features and metabolic risk highlights their potential role as visible indicators of underlying dysfunction and aids early identification of high-risk PCOS phenotypes.[27]

Study Limitations

This study had a few limitations. Its cross-sectional design restricts causal inferences between predictor variables and metabolic outcomes, underscoring the need for prospective longitudinal studies. Residual confounding from unmeasured factors such as diet, physical activity, and supplement use cannot be excluded, particularly in relation to micronutrient status. As the cohort was urban, the findings may not be generalizable to all Indian women with PCOS due to variations in genetics, comorbidities, and healthcare access. Larger, multicenter prospective studies with long-term follow-up are required for more definitive conclusions.

Conclusion

Our findings underscore the pivotal role of early metabolic screening in women with PCOS to enable timely weight management, lifestyle modification, and prevention of long-term complications. Incorporating micronutrient assessment into routine evaluation offers a simple, low-cost, and pragmatic screening tool, particularly relevant in resource-sensitive settings. Early correction of nutritional deficiencies can mitigate obesity-related metabolic risks, reduce healthcare costs, and enhance productivity, while improving overall reproductive and metabolic health. At the community level, such integrated approaches may significantly reduce the burden of PCOS, and at the national and global level, they present a scalable preventive health strategy with the potential to strengthen women's health outcomes worldwide.

Orcid

Zubeda Tumbi https://orcid.org/0009-0004-4915-3136

References

1. Jurczewska J, Ostrowska J, Chełchowska M, et al. Abdominal obesity in women with polycystic ovary syndrome and its relationship with diet, physical activity, and insulin resistance: a pilot study. Nutrients 2023;15(16):3652.
2. Legro RS, Castracane VD, Kauffman RP. Detecting insulin resistance in polycystic ovary syndrome: purposes and pitfalls. Obstet Gynecol Surv 2004;59(2):141–154.
3. Marshall JC, Dunaif A. Should all women with PCOS be treated for insulin resistance? Fertil Steril 2012;97(1):18–22.
4. Fahed G, Aoun L, Zerdan MB, et al. Metabolic syndrome: updates on pathophysiology and management in 2021. Int J Mol Sci 2022;23(2):786.
5. Manzano-Nunez R, Santana-Dominguez M, Rivera-Esteban J, 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.

6. Fazleen NE, Whittaker M, Mamun A. Risk of metabolic syndrome in adolescents with polycystic ovarian syndrome: a systematic review and meta-analysis. Diabetes Metab Syndr 2018;12(6):1083–1090.
7. Lim SS, Kakoly NS, Tan JWJ, et al. Metabolic syndrome in polycystic ovary syndrome: a systematic review, meta-analysis and meta-regression. Obes Rev 2019;20(2):339–352.
8. Otaghi M, Azami M, Khorshidi A, et al. The association between metabolic syndrome and polycystic ovary syndrome: a systematic review and meta-analysis. Diabetes Metab Syndr 2019;13(2):1481–1489.
9. Khorshidi A, Azami M, Tardeh S, et al. The prevalence of metabolic syndrome in patients with polycystic ovary syndrome: a systematic review and meta-analysis. Diabetes Metab Syndr 2019;13(4):2747–2753.
10. Zaeemzadeh N, Sadatmahalleh SJ, Ziaei S, et al. Comparison of dietary micronutrient intake in PCOS patients with and without metabolic syndrome. J Ovarian Res 2021;14(1):10.
11. Wang J, Wang B, Li C, et al. Evolving global trends in PCOS burden: a three-decade analysis (1990–2021) with projections to 2036 among adolescents and young adults. Front Endocrinol 2025;16:1569694.
12. Shukla A, Rasquin LI, Anastasopoulou C. Polycystic ovarian syndrome. In: StatPearls [Internet]. StatPearls Publishing; 2025.
13. Dokras A. Polycystic ovary syndrome in 2025—insights and innovations. Fertil Steril 2025;124(5 Pt 2):907–909.
14. Christ JP, Cedars MI. Current guidelines for diagnosing PCOS. Diagnostics 2023;13(6):1113.
15. Williams J, Walton S, Wells J. Standard Operating Procedures: Adult Anthropometry (Life Study Working Papers). Life Course Epidemiology and Biostatistics/UCL Institute of Child Health; 2016.
16. World Health Organization. Waist Circumference and Waist–Hip Ratio: Report of a WHO Expert Consultation. Geneva: WHO Press; 2008.
17. Vargas-Uricoechea H, Nogueira JP, Pinzón-Fernández MV, et al. Population status of vitamin B12 values in the general population and in individuals with type 2 diabetes, in Southwestern Colombia. Nutrients 2023;15(10):2357.
18. Camacho PM, Petak SM, Binkley N, et al. American Association of Clinical Endocrinologists/American College of Endocrinology clinical practice guidelines for the diagnosis and treatment of postmenopausal osteoporosis—2020 update. Endocr Pract 2020;26(Suppl 1):1–46.
19. Baş H, Filiz S. Evaluation of ovarian stromal microvascularity and clinical-hormonal associations in reproductive-aged women with polycystic ovary morphology. Diagnostics (Basel) 2025;15(11):1376.
20. Benjamini Y, Hochberg Y. Controlling the false discovery rate: a practical and powerful approach to multiple testing. J R Stat Soc Ser B Methodol 1995;57(1):289–300.
21. Misra A, Vikram NK, Gupta R, et al. Waist circumference cutoff points and action levels for Asian Indians for identification of abdominal obesity. Int J Obes 2006;30(1):106–111.
22. Agarwal M, Sinha S, Lohani P, et al. Polycystic ovarian syndrome in aging women: an observational study. Cureus 2022;14(9):e29776.
23. Gautam PK, Saxena P, Tripathi S, et al. Comparative analysis of serum fetuin—a levels in women with PCOS and controls. Eur J Cardiovasc Med 2025;15:174–178.
24. Amisi CA. Markers of insulin resistance in polycystic ovary syndrome women: an update. World J Diabetes 2022;13(3):129–149.
25. Mansour A, Mirahmad M, Mohajeri-Tehrani MR, et al. Risk factors for insulin resistance related to polycystic ovarian syndrome in Iranian population. Sci Rep 2023;13:10269.
26. Singh N, Hooja N, Yadav A, et al. Waist circumference and waist-to-hip ratio as indicators of abdominal obesity. Int J Life Sci Res Arch 2022;2(2):102–105.
27. Soares EMM, Azevedo GD, Gadelha RGN, et al. Prevalence of the metabolic syndrome and its components in Brazilian women with polycystic ovary syndrome. Fertil Steril 2008;89(3):649–655.
28. Sharma D, Sharma S, Verma A, et al. PCOS (polycystic ovary syndrome) and its correlation with waist circumference, glucose metabolism, and lipid profile. Int J Pharm Clin Res 2022;14(8):560–564.
29. Sun Y, Wang W, Shen Q, et al. Waist circumference coupled with either HDL-C or TG can be used as a diagnostic marker for metabolic syndrome in Chinese women with polycystic ovary syndrome. Int J Endocrinol 2018;2018:6102085.
30. Pazderska A, Tun TK, Phelan N, et al. In women with PCOS, waist circumference is a better surrogate of glucose and lipid metabolism than disease status per se. Clin Endocrinol 2018;88(4):565–574.
31. Giri A, Joshi A, Shrestha S, et al. Metabolic syndrome among patients with polycystic ovarian syndrome presenting to a tertiary care hospital: a descriptive cross-sectional study. JNMA J Nepal Med Assoc 2022;60(246):137–141.
32. Gudiseva VSH, Gurupadappa K, Manjula B. Correlation of dyslipidemia with HbA1c, fasting plasma glucose, and BMI in patients with PCOS and healthy women. Al Ameen J Med Sci 2024;17(2):109–114.
33. Saravia G, Civeira F, Hurtado-Roca Y, et al. Glycated hemoglobin, fasting insulin, and the metabolic syndrome in males. Cross-sectional analyses of the Aragon Workers' Health Study baseline. PLoS One 2015;10(8):e0132244.
34. Carrasco-Cabezas M, Assmann TS, Martínez P, et al. Folate and vitamin B12 levels in Chilean women with PCOS and their association with metabolic outcomes. Nutrients 2024;16(12):1937.
35. Ulloque-Badaracco JR, Al-Kassab-Córdova A, Hernández-Bustamante EA, et al. Homocysteine, vitamin B12, and folate circulating levels in women with and without polycystic ovary syndrome: a systematic review and meta-analysis. Women's Health 2024;20:17455057241279039.
36. Su N, Li J, Xia Y, et al. Non-causal relationship of polycystic ovarian syndrome with homocysteine and B vitamins: evidence from a two-sample Mendelian randomization. Front Endocrinol 2024;15:1393847.
37. Białka-Kosiec A, Orszulak D, Gawlik A, et al. The relationship between the level of vitamin D, leptin, and FGF23 in girls and young women with polycystic ovary syndrome. Front Endocrinol 2022;13:1000261.
38. Kim JJ, Choi YM, Chae SJ, et al. Vitamin D deficiency in women with polycystic ovary syndrome. Clin Exp Reprod Med 2014;41(2):80–85.
39. Shen JY, Xu L, Ding Y, et al. Effect of vitamin supplementation on polycystic ovary syndrome and key pathways implicated in its development: a Mendelian randomization study. World J Clin Cases 2023;11(23):5468–5478.