Original Article

Body Composition, Metabolic Characteristics, and Insulin Resistance in Obese and Nonobese Women with Polycystic Ovary Syndrome

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Abstract

Objectives: The objective was to compare body composition, metabolic characteristics, and insulin resistance between obese (body mass index [BMI] ≥25 kg/m^2) polycystic ovary syndrome (PCOS) and nonobese PCOS (BMI <25 kg/m^2) women and their age- and BMI-matched controls. **Materials and Methods:** A total of 81 PCOS women (Rotterdam criteria) (obese – 42; nonobese – 39) and 86 controls (obese – 42; nonobese –44) were recruited in this cross-sectional study. All women underwent a detailed assessment of clinical, anthropometric, and metabolic parameters, insulin resistance indices, and body composition measurements with visceral adipose tissue assessment (VAT) (dual-energy X-ray absorptiometry scan). **Results:** Of PCOS women, 27% (80% – obese PCOS; 20% – nonobese PCOS) were diagnosed with metabolic syndrome (International Diabetes Federation criteria), 35% of PCOS women (46% – obese PCOS; 54% – nonobese PCOS) had impaired glucose tolerance, and 7% of PCOS women (2/3rd – obese PCOS; 1/3rd – nonobese PCOS) had diabetes mellitus. Insulin resistance was seen in about 80% in obese PCOS women and 20% in nonobese PCOS women based on various insulin resistance indices such as fasting insulin (≥12.2 μU/ml), Homeostasis Model Assessment-Insulin Resistance (≥2.5), and Quantitative Insulin Sensitivity Check Index (<0.33). Total body fat, estimated (Est.) VAT, and corrected Est. VAT (corrected for body weight) were significantly increased (P = 0.0001) in both obese and nonobese PCOS women when compared to those of their age- and BMI-matched controls. However, corrected Est. VAT (corrected for body weight) was not significantly different between obese and nonobese PCOS women. **Conclusion:** Both obese and nonobese PCOS women when compared with their age- and BMI-matched controls were metabolically worse and had more visceral adiposity. Nonobese PCOS poses similar risk as that of obese PCOS in having similar amount of VAT (corrected for body weight).

Keywords: *Insulin resistance, nonobese polycystic ovary syndrome, obese polycystic ovary syndrome, polycystic ovary syndrome, visceral adipose tissue*

Introduction

Polycystic ovary syndrome (PCOS) is one of the most common hormonal disorders in women of reproductive age. Its prevalence has been reported to range from 13% to 22% in South Asian studies[1-3] and between 4% and 11% in Western literature.[4] The consequences of PCOS are beyond the reproductive axis with substantial risk for the development of metabolic disturbances and cardiovascular abnormalities similar to metabolic syndrome.[5,6] This is common because both PCOS and metabolic syndrome share insulin resistance

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as a central pathognomic feature. Hence, PCOS might be viewed as a gender-specific form of metabolic syndrome, and the term "syndrome X" has been suggested for the same.[7] When compared with other populations, South Asians seem to have a greater propensity for developing metabolic syndrome, the prevalence of which is about two-thirds in patients with PCOS.[8-10]

Abnormal fat distribution, particularly visceral adipose tissue (VAT), and insulin resistance are the key features of PCOS.[11] There are conflicting results with respect to abnormal fat distribution and metabolic profile among lean PCOS and obese PCOS women in various Western and South Asian studies. Some studies showed significant differences among this subset of PCOS women,[12,13] whereas others did not show significant differences.[14]

However, it is not clear whether the lean PCOS women have similar pattern of metabolic abnormalities and fat distribution as that of obese PCOS women. It is also not clear whether lean PCOS women are at similar risk of developing metabolic abnormalities as that of their obese counterparts. There is also a paucity of information with regard to body composition, metabolic parameters, and insulin resistance among South Asian lean and obese PCOS women in comparison with their age- and body mass index (BMI)-matched controls. Hence, this study was aimed at studying the body composition, metabolic characteristics, and insulin resistance in PCOS women in comparison with their age- and BMI-matched controls which will help in better understanding of the abnormal metabolic profile in PCOS women from a Indian perspective.

Materials and Methods

Subjects

This study was conducted over a period of 20 months from August 1, 2016, to March 31, 2018, at a tertiary care center in southern part of India. Women with PCOS attending the Endocrinology and Reproductive Medical Unit clinic were classified as "cases." Healthy women without PCOS from the community were considered as "controls" for comparison with cases. Written informed consent was taken from all the women enrolled in the study. The study protocol was approved by the institutional review board.

All women recruited in the study fulfilled the inclusion criteria. Women who were 18–35 years of age with phenotypic features of PCOS and fulfilling the Rotterdam criteria[15] for the diagnosis of PCOS were included in the study. Secondary causes of oligo-anovulation (hyperprolactinemia, Cushing's syndrome, untreated hypothyroidism, congenital adrenal hyperplasia, and adrenal tumors) were excluded by appropriate tests. Women who had a history of intake of drugs which can cause hirsutism (androgens, valproic acid, cyclosporine, diazoxide, or minoxidil) or a history of intake of drugs which can alter body the composition (oral contraceptive pills, metformin, thiazolidinediones, or spironolactone for more than the preceding 3 months) or pregnancy were excluded from the study. Women with preexisting diabetes mellitus before the diagnosis of PCOS were also excluded from the study.

Study design

This was a cross-sectional study wherein all the recruited women were divided into four groups according to BMI[16,17] as follows: obese PCOS (BMI ≥25 kg/m^2); nonobese PCOS (BMI <25 kg/m^2); obese controls (BMI ≥25 kg/m^2); and nonobese controls (BMI <25 kg/m^2). All women underwent detailed history taking and physical examination. Irregular menstrual cycles in the form of oligomenorrhea/amenorrhea, hirsutism, infertility, family history (diabetes mellitus/PCOS/hypertension), detailed dietary intake, and data regarding physical activity by Global Physical Activity Questionnaire were all noted. All women underwent a detailed assessment including anthropometric measurements (height, weight, BMI, and waist–hip ratio), blood pressure, hirsutism assessment (modified Ferriman–Gallwey score; score ≥8 is significant), and signs of insulin resistance such as acanthosis nigricans and skin tags.

All women underwent the following biochemical tests: fasting blood glucose, 2 h post 75 g blood glucose (enzymatic method), hemoglobin A1c (high-performance liquid chromatography), fasting lipid profile (enzymatic colorimetric assay-oxidase peroxidase method), fasting insulin (chemiluminescence immunoassay), 8-am total testosterone (chemiluminescence immunoassay), free testosterone, sex hormone-binding globulin (SHBG), and dehydroepiandrosterone sulfate (chemiluminescence immunoassay). All women also underwent biochemical tests (prolactin, thyroid-stimulating hormone, 17-hydroxyprogesterone, and post 1-mg dexamethasone serum cortisol) for the exclusion of secondary causes of PCOS. Various insulin resistance indices[18-22] were used for assessing insulin resistance namely fasting insulin level (cutoff value; ≥12.2 μU/ml), Homeostasis Model Assessment-Insulin Resistance (HOMA-IR) (cutoff value; ≥2.5), and Quantitative Insulin Sensitivity Check Index (QUICKI) (cutoff value; <0.33). Whole-body dual-energy X-ray absorptiometry (DXA) scan using Hologic DXA QDR 4500 Discovery A

machine with a coefficient of variation (CV) of 2% was used for the study of body composition and estimation of VAT in all women (both cases and controls).

All the parameters including anthropometric, metabolic characteristics, various insulin resistance indices, and body composition parameters along with VAT were compared between PCOS women and their age- and BMI-matched controls.

Sample size calculation

The sample size was calculated based on the data published by Remsberg *et al*.[23] With the consideration of 4% body fat difference between the two groups (obese PCOS and nonobese PCOS), 80% power, and alpha error of 5%, the sample size required was 36 in each group. An equal number of patients were required in each of the control groups (obese controls and nonobese controls) for comparison between cases and controls.

Statistical analysis

Statistical analysis was done using SPSS for Windows, Version 16.0. (SPSS Inc., Chicago, USA). Two-sample *t*-test was used to compare the mean differences in continuous outcome variables between the groups. Similarly, the Chi-square test was used for the association between the categorical variables, as appropriate. Logistic univariate regression analysis was used for predicting the risk of metabolic syndrome among the various parameters (free testosterone index, estimated [Est]. VAT, and corrected Est. VAT). Spearman's and Pearson's correlations were used for nonparametric and parametric correlations, respectively.

Results

A total of 81 PCOS women (obese – 42; nonobese – 39) and 86 controls (obese – 42; nonobese – 44) were recruited in the study. All women had oligo-anovulation and sonological appearance of polycystic ovaries (100%); hyperandrogenism either biochemical or clinical was seen in around 45% of PCOS women (obese – 60%; nonobese – 30%). Demographic and other baseline characteristics of PCOS women and controls are shown in Table 1. Around 27% of PCOS women (80% – obese PCOS; 20% – nonobese PCOS) were diagnosed with metabolic syndrome (International Diabetes Federation [IDF] criteria). Impaired glucose tolerance was seen in 35% of PCOS women (46% – obese PCOS; 54% – nonobese PCOS) and about 7% of PCOS women (2/3rd – obese PCOS; 1/3rd –nonobese

Table 1: Baseline characteristics of polycystic ovary syndrome women (obese and nonobese) and controls (obese and nonobese)

Variables (units and normal range)	Obese group (BMI ≥25 kg/m^2)			Nonobese group (BMI <25 kg/m^2)		
	PCOS (*n*=42), mean±SD	Controls (*n*=42), mean±SD	*P*	PCOS (*n*=39), mean±SD	Controls (*n*=44), mean±SD	*P*
Baseline parameters						
Age (years)	25.2±3.9	25.3±3.8	0.89	25.4±3.8	24.1±4.4	0.13
Height (cm)	155.1±5.7	154.9±6.9	0.87	154.9±4.6	154.9±6.2	0.95
Weight (kg)	74.3±12.2	71.1±11.9	0.23	53.4±6.7	51.3±6.6	0.15
BMI (kg/m^2)	30.9±4.9	29.6±4.4	0.22	22.2±2.4	21.4±2.7	0.13
Waist (cm)	94.7±9.6	91.7±11.7	0.19	82.6±15.4	72.3±8.1	0.0001
Hip (cm)	103.7±10.2	104.2±9.8	0.91	93.9±12.1	86.3±7.8	0.001
Waist-hip ratio	0.9±0.1	0.8±0.8	0.05	0.9±0.1	0.8±0.6	0.08
GPAQ (METS/week)	2.2±0.1	2.4±0.5	0.11	2.2±0.5	2.3±0.4	0.41
Biochemical parameters of hyperandrogenism						
SHBG (26.1-110 nmol/L)	27.1±16.5	44.2±19.2	0.0001	46.2±28.3	58.3±30.15	0.06
	22.4 (7.2-76)‡	38.6 (7.6-108)‡		38.6 (17.3-151)‡	56.9 (4.4-156)‡	
Total testosterone (50-120 ng/dL)	40.4±46.6	34.3±28.1	0.47	32.3±24.8	27.2±14.6	0.27
	25.7 (20-298)‡	4.3 (1.1-21.2)‡		20 (0.9-150)‡	20 (0.8-78)‡	
Free testosterone index (0.51%-6.53%)	5.7±4.7	5.9±14.1	0.95	2.9±2.2	2.4±2.8	0.28
	4.3 (1.1-21.2)‡	2.5 (0.9-86)‡		2.7 (0.4-12.3)‡	1.3 (0.4-15.8)‡	
Free testosterone (0.76%-2.06%)	2.4±1.7	4.5±18.3	0.46	1.5±0.4	1.3±0.6	0.06
	2.1 (1-12.4)‡	1.6 (1-119)‡		1.5 (0.6-2.3)‡	1.2 (0.5-3.4)‡	
DHEAS (35-430 µg/dL)	116.3±125.8	131.9±65.4	0.48	110.2±55.7	141.6±105.6	0.09
	83.7 (18.7-785)‡	118 (38-311)‡		101 (1.6-232)‡	134 (15-648)‡	

‡Median (minimum-maximum). PCOS=Polycystic ovary syndrome, BMI=Body mass index, GPAQ=Global Physical Activity Questionnaire, METS=Metabolic equivalent tasks, SHBG=Sex hormone-binding globulin, DHEAS=Dehydroepiandrosterone sulfate, SD=Standard deviation

PCOS) had diabetes mellitus. Insulin resistance was seen in 80% of obese PCOS women and 20% of nonobese PCOS women based on various insulin resistance indices such as fasting insulin (≥12.2 μU/ml), HOMA-IR (≥2.5), and QUICKI (<0.33). Comparison of metabolic parameters, insulin resistance indices, and body composition parameters among PCOS women with their age- and BMI-matched controls is shown in Table 2. A comparison was also done with respect to these parameters among obese and nonobese PCOS women which is shown in Table 3.

Total body fat and truncal fat were statistically significantly higher (P = 0.0001) in PCOS women when compared to their age- and BMI-matched controls. PCOS women (both obese and nonobese) had an increased Est. VAT (assessed by DXA scan) which was statistically significant (P = 0.0001) when compared to that of their age- and BMI-matched controls, and the difference was significant even after correcting for their body weight. However, Est. VAT adjusted for body weight (corrected Est. VAT) was not significantly different between obese and nonobese PCOS women, suggesting that nonobese PCOS women have similar amount of VAT as that of obese PCOS when adjusted for their body weight. In a univariate logistic regression analysis, various parameters were assessed for the risk of development of metabolic syndrome [Table 4]. Est. VAT volume and corrected Est. VAT volume (corrected for body weight) significantly predicted the risk of metabolic syndrome. SHBG levels showed statistically significant negative correlation with HOMA-IR (P < 0.001) [Figure 1].

Discussion

This study attempted to evaluate the metabolic characteristics, insulin resistance indices, and body composition parameters in obese and nonobese PCOS women in comparison to their age- and BMI-matched controls.

Nonobese and obese PCOS women when compared to their age- and BMI-matched controls had higher total body fat, truncal fat, and Est. VAT (assessed by DXA scan). Corrected Est. VAT was not significant between

Table 2: Comparison of metabolic parameters, insulin resistance indices, and body composition parameters between polycystic ovary syndrome women (obese and nonobese) and controls (obese and nonobese)

Variables (units and normal range)	Obese group (BMI ≥25 kg/m²)			Nonobese group (BMI <25 kg/m²)		
	PCOS (*n*=42), mean±SD	Controls (*n*=42), mean±SD	*P*	PCOS (*n*=42), mean±SD	Controls (*n*=42), mean±SD	*P*
Metabolic parameters						
Fasting glucose (70-100 mg/dL)	97.8±14.7	90.5±15.1	0.03	94.7±17.9	85.8±6.4	0.005
2 h post 75 g blood glucose (<140 mg/dL)	121.7±41.3	111.8±46.9	0.33	117.9±38.4	103.8±29.3	0.07
HbA1c (<5.7%)	5.6±0.7	5.5±0.9	0.84	5.45±0.9	5.2±0.3	0.11
Total cholesterol (<200 mg/dL)	173.6±33.2	157.9±25.9	0.02	175.6±33.6	147.2±30	0.0001
Triglycerides (<150 mg/dL)	88±43.3	92±46.6	0.69	80±31.6	73.4±36.7	0.38
HDL-cholesterol (>60 mg/dL)	44.3±8.9	40.9±9.8	0.09	50.4±10.8	42.4±9.9	0.001
LDL-cholesterol (<100 mg/dL)	109.4±29.9	103.6±22.3	0.32	107.8±27.8	93±25.3	0.01
Non-HDL cholesterol (mg/dL)	129.3±34.9	111.2±27.8	0.11	125.2±34.4	104.1±27.7	0.003
Insulin resistance indices						
Fasting insulin (0-30 μU/ml)	26.1±44.5	10.5±10.3	0.03	8.58±3.8	6.4±3.7	0.01
	16.3 (6-290)‡	9.7 (0.7-65.2)‡		7.8 (2-19.3)‡	5.4 (2-18)‡	
HOMA-IR (≥2.5)	7.2±16	2.5±2.9	0.06	2.1±1.2	1.3±0.83	0.004
	3.8 (1.1-105.2)‡	2 (0.16-18.67)‡		1.8 (0.4-6.6)‡	1.2 (0.3-4.4)‡	
QUICKI (<0.33)	0.31±0.03	0.39±0.25	0.04	0.35±0.03	0.38±0.04	0.001
	0.3 (0.22-0.38)‡	0.3 (0.3-0.5)‡		0.3 (0.3-0.5)‡	0.4 (0.3-1.9)‡	
Body composition parameters						
Total body fat (%)	42.2±3.8	35.5±4.9	0.0001	37.9±4.7	30.5±5.5	0.0001
Fat trunk/fat legs (%)	0.9±0.1	0.8±0.1	0.0001	0.8±0.1	0.7±0.1	0.0001
Estimated VAT volume (cm^3)	450.9±150.6	286.1±137.3	0.0001	309.6±120.7	176.2±86.9	0.0001
Corrected estimated VAT volume to body weight (cm^3/kg)	6.11±1.91	4.16±2.15	0.0001	5.74±1.91	3.58±1.62	0.0001
Fat mass (g)	31,701±7254	31,991±41,648	0.96	20,424±4603	16,064±3862	0.0001
Total mass (g)	74,654±11,945	70,664±12,392	0.14	53,854±6585	49,604±7406	0.008

‡Median (minimum-maximum). PCOS=Polycystic ovary syndrome, BMI=Body mass index, HDL=High-density lipoprotein, LDL=Low-density lipoprotein, HOMA-IR=Homeostatic Model Assessment-Insulin Resistance, QUICKI=Quantitative Insulin Sensitivity Check Index, VAT=Visceral adipose tissue, HbA1c=Glycated hemoglobin, SD=Standard deviation

Table 3: Comparison of insulin resistance indices, metabolic parameters, and body composition parameters between obese polycystic ovary syndrome and nonobese polycystic ovary syndrome

Variables (units and normal range)	Mean±SD		*P*
	Obese PCOS (BMI ≥25 kg/m²) (*n*=42)	Nonobese PCOS (BMI <25 kg/m²) (*n*=39)	
Metabolic parameters			
Glucose fasting (70-100 mg/dL)	97.8±14.7	94.7±17.9	0.39
2 h post 75 g blood glucose (<140 mg/dL)	121.7±41.3	117.9±38.4	0.67
HbA1c (<5.7%)	5.6±0.7	5.45±0.9	0.53
Total cholesterol (<200 mg %)	173.6±33.2	175.6±33.6	0.79
Triglycerides (<150 mg %)	88±43.3	80±31.6	0.35
HDL-cholesterol (>60 mg %)	44.3±8.9	50.4±10.8	0.009
LDL-cholesterol (<100 mg %)	109.4±29.9	107.8±27.8	0.79
Non-HDL cholesterol	129.3±34.9	125.2±34.4	0.61
Insulin resistance indices			
Fasting insulin (0-30 µU/ml)	26.1±44.5	8.58±3.8	0.01
	16.3 (6-290)‡	7.8 (2-19.3)‡	
HOMA-IR (≥2.5)	7.2±16	2.1±1.2	0.05
	3.8 (1.1-105.2)‡	1.8 (0.4-6.6)‡	
QUICKI (>0.33)	0.31±0.03	0.35±0.03	0.001
	0.3 (0.22-0.38)‡	0.3 (0.3-0.5)‡	
Body composition parameters			
Total body fat (%)	42.2±3.8	37.9±4.7	0.0001
Fat trunk/fat legs (%)	0.9±0.07	0.85±0.12	0.008
Estimated VAT volume (cm³)	450.9±150.6	309.6±120.7	0.0001
Corrected estimated VAT volume to body weight (cm³/kg)	6.11±1.91	5.74±1.91	0.39
Fat mass (g)	31,701±7254	20,424±4603	0.0001
Total mass (g)	74,654±11,945	53,854±6585	0.0001

‡Median (minimum-maximum). PCOS=Polycystic ovary syndrome, BMI=Body mass index, HDL=High-density lipoprotein, LDL=Low-density lipoprotein, HOMA-IR=Homeostatic Model Assessment-Insulin Resistance, QUICKI=Quantitative Insulin Sensitivity Check Index, VAT=Visceral adipose tissue, HbA1c=Glycated hemoglobin, SD=Standard deviation

Table 4: Logistic regression analysis: Metabolic syndrome in polycystic ovary syndrome women

Variables	Metabolic syndrome		
	Relative risk	*P*	95% CI
Free testosterone index (>6.53%)	0.25	2.10	0.59-7.50
Estimated VAT volume (393 cm³)	10.85	<0.001	2.87-41.10
Corrected estimated VAT volume (5.7 cm³/kg)	7.16	0.001	2.14-23.95

VAT=Visceral adipose tissue, CI=Confidence interval

obese and nonobese PCOS women suggesting that nonobese PCOS women had similar amount of VAT as that of obese PCOS women when adjusted for their body weight. Overall, the present study suggests that PCOS women, irrespective of BMI, have increased visceral adiposity which predisposes them to a higher risk for future development of metabolic complications. The literature is not conclusive with respect to abdominal obesity (as assessed by either DXA scan or magnetic resonance imaging [MRI]) in PCOS women

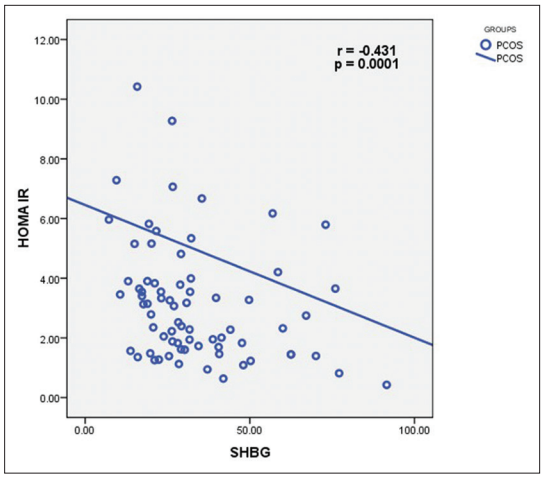

Figure 1: Correlation between sex hormone-binding globulin and Homeostasis Model Assessment-Insulin Resistance

when compared to controls. Some studies demonstrated higher VAT in PCOS women than that of age- and BMI-matched controls;[12,13] however, other studies did not find any statistically significant difference with regard to VAT in PCOS women compared to age- and

BMI-matched controls.[14,24] Limited literature is available in this regard from an Indian perspective.[13] Visceral fat estimation either indirectly by waist–hip ratio or more objectively by DXA scan and MRI, is an important determinant and a surrogate marker for insulin resistance as this will predispose PCOS women to high risk of developing metabolic complications by various mechanisms.[25,26]

In the present study, insulin resistance was seen in about 75%–80% of obese PCOS women and in about 18%–20% of nonobese PCOS women, based on fasting insulin cutoff value of ≥12.2 μU/mL,[18,19] HOMA-IR of ≥2.5,[18,20-22] and QUICKI index of <0.33[18,21,22] for determining insulin resistance. Fasting insulin and QUICKI values were statistically significantly higher ($P < 0.05$) in both groups of PCOS women (obese and nonobese) when compared with their age- and BMI-matched controls. Nonobese PCOS women compared with nonobese controls were metabolically worse and had greater fat-corrected insulin resistance than the obese PCOS women compared with obese controls. Thus, the findings in the present study suggest that the nonobese PCOS group (overweight and normal weight PCOS) were less insulin resistant when compared to the obese PCOS group. However, both groups (obese and nonobese PCOS) have similar amount of VAT (when corrected for body weight) as assessed by DXA scan. Hence, we postulate that there may be factors other than insulin resistance which make the nonobese PCOS women gain more weight and VAT. Some studies have shown that there may be an alteration in satiety which can lead to excess eating, causing obesity in women with PCOS.[27-29] There may be associated factors such as meal-stimulated decreased glucagon-like peptide-1 levels[28] and decreased meal-stimulated cholecystokinin levels[29] which lead to alteration in satiety. Various environmental and genetic factors along with the above mechanisms may further contribute to more abdominal and visceral adiposity even in PCOS women with normal weight.

A total of 22 out of 81 (27%) PCOS women (80% – obese PCOS; 20% – overweight PCOS) had a diagnosis of metabolic syndrome based on the IDF criteria.[30] Around 2/5th of the PCOS women had features of either clinical or biochemical evidence of hyperandrogenism. Women with features of hyperandrogenism represent a more severe phenotype of PCOS, which is metabolically unhealthy because hyperandrogenism has also been related to obesity.[31]

In a univariate logistic regression analysis, Est. VAT volume (relative risk [RR]: 10.8; 95% confidence interval [CI]: 2.8–41.1) and corrected Est. VAT volume to body weight (RR: 7.1; 95% CI: 2.1–23.9) were better predictors among the various parameters, for the risk of metabolic syndrome.

Our study has several merits. It is the first Indian study, which has analyzed body composition (assessed by DXA scan) in obese PCOS and nonobese PCOS women (overweight and normal weight) and compared it with their age- and BMI-matched controls. The metabolic characteristics and body composition parameters along with VAT were studied separately in the subset of PCOS women namely obese PCOS and nonobese PCOS. This will provide better understanding of the metabolic disturbances in PCOS. However, there were a few limitations. A BMI cutoff of <25 kg/m^2 was considered for nonobese group which includes both normal weight women (BMI <23 kg/m^2) and overweight women (BMI ≥23 kg/m^2 but ≤25kg/m^2) due to the inadequate number in the normal weight PCOS group. Ideally, there should have been three groups (obese, overweight, and normal weight) in both PCOS and control groups with adequate number for proper assessment and better interpretation as follows: normal weight PCOS and controls (BMI <23 kg/m^2); overweight PCOS and controls (BMI ≥23 kg/m^2 but ≤25 kg/m^2); and obese PCOS and controls (BMI >25 kg/m^2). Insulin resistance was studied by surrogate markers of insulin resistance such as fasting insulin, HOMA-IR, and QUICKI, which may reflect more of hepatic insulin resistance. More sophisticated studies such as hyperinsulinemic euglycemic clamps would have helped in better assessment of insulin resistance.

Conclusion

Obese and nonobese PCOS women when compared with their age- and BMI-matched controls were metabolically worse and had more visceral adiposity. Nonobese PCOS women pose similar risk as that of obese PCOS women in having similar amount of VAT (corrected for body weight) with the risk of development of metabolic complications. Nonobese PCOS should be managed on similar lines as that of obese PCOS for the prevention of metabolic complications in future.

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Conflicts of interest

There are no conflicts of interest.

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