

POLYCYSTIC OVARIAN SYNDROME AND ITS RELATIONSHIP WITH OBESITY AMONG ASSAMESE WOMEN IN ASSAM, NORTHEAST INDIA

CHUMI DAS¹, JUMI DAS², TILUTTOMA BARUAH³,
NITISH MONDAL⁴

¹Department of Anthropology, Cotton University, Guwahati, India

²Department of Zoology, Cotton University, Guwahati, India

³Department of Humanities and Social Sciences, Assam Down Town University, Guwahati, India

⁴Department of Anthropology, School of Human Sciences, Sikkim University, Gangtok, Sikkim, India

ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common endocrinological disorder affecting 6–10% of women in their reproductive years and causing various metabolic syndromes. PCOS is often confused with coexisting obesity, insulin resistance, and metabolic syndrome. Obesity is common in PCOS and affects between 30–70% of reproductive women, depending on the study setting and the ethnic background of the research participants.

Objective: To study the prevalence of obesity in PCOS women and its impact on various biological parameters associated with PCOS.

Materials and methods: A hospital-based prospective study was undertaken, which included a sample of 100 reproductive women diagnosed with PCOS. Anthropometric and various biological variables were obtained for the present study.

Results: The results showed that obese PCOS women constituted 71.00% and non-obese PCOS women 29.00% of the subjects. The biological variables were found to have a higher impact among obese PCOS women. Menstrual irregularity (12.68%) and hypertension (5.63%) were significantly higher in obese PCOS women. The anthropometric indices also played a significant role among the obese PCOS women, and the findings showed that the body mass

index (BMI; $26.20 \pm 2.70 \text{ kg/m}^2$) and waist-hip ratio (WHR; $0.90 \pm 0.32 \text{ kg/m}^2$) showed a high predictive value.

Conclusion: Obese PCOS women have a higher risk of adverse fertility and metabolic outcomes. The anthropometric parameters were found to be strongly associated with obesity among PCOS women.

Keywords: *polycystic ovarian syndrome; obesity; hyperandrogenism; anthropometry*

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common endocrinological disorder affecting 6–10% of women in their reproductive years [1]. It is associated with a significantly higher odds ratio for the development of various cardiovascular risk factors [2–6]. The cause of PCOS remains unknown; however, its increased prevalence coincides with a global increase in the incidence of the metabolic syndrome [1, 4–6]. Metabolic disturbance is associated with increased adiposity and clinically worsens the menstrual cycle, hyper-androgenism, ovulation rates, infertility, maternal and neonatal outcomes, and increases the risks for diabetes, cardiovascular disease, and cancer in women [4–7]. Despite the high prevalence and significant morbidity resulting from both reproductive and hyper-androgenic features and the associated cardiovascular risks, the etiology of PCOS remains incompletely understood [6–10].

The National Institutes of Health (NIH) sponsored a consensus conference in 1990 that made the first attempt to define PCOS [11]. The Rotterdam criteria [12] defined PCOS by including three disorders: oligo or anovulation, clinical and/or biochemical signs of hyper-androgenism, and polycystic ovaries. In essence, the Rotterdam criteria [12] expanded the NIH 1990 definition [13]. The Androgen Excess Society's (AES) position statement [13] recommended the most recent modification of the diagnostic criteria for PCOS. The AES is an internationally recognized organization dedicated to promoting knowledge and research in all aspects of androgen excess disorders. The panel of experts appointed by AES concluded that the characteristic associated with PCOS is metabolic risk, particularly insulin resistance. Keeping this in mind, they assessed all of the published large-scale studies on the PCOS phenotype [4–7, 14].

Polycystic ovary syndrome is a heterogeneous condition associated with features of the metabolic syndrome. Consequently, several studies involving women with PCOS are often confounded by coexisting obesity, insulin resis-

tance, and other features of the metabolic syndrome [4–8]. Obesity is common in PCOS and affects between 30–70% of women, depending on the setting of the study and the ethnic background of the research participants [8, 15–17]. Many women with PCOS (38%–88%) are overweight or obese [14, 18–20]. Obesity further augments the adverse metabolic and reproductive outcomes of PCOS [16, 17, 20]. Obesity increases insulin resistance and compensatory hyper-insulinemia, glucose intolerance, dyslipidemia, and the risk of pregnancy complications [4, 5, 16, 21]. Obese women with PCOS have a more severe phenotype than those who are less obese, with more severe menstrual irregularity, infertility, miscarriage, hypertension, gestational diabetes, prematurity, biochemical and clinical hyper-androgenism, glucose intolerance and/or type-2 diabetes, and metabolic syndrome [20–24]. However, not all obese women develop PCOS: some primary abnormality in androgen production is supposed to be necessary for the development of the syndrome [10, 25, 26]. The present investigation aims to find the prevalence of PCOS in reproductive women and compare it between the two PCOS phenotypes: obese and non-obese PCOS groups.

MATERIAL AND METHODS

A prospective study was conducted in the Department of Gynecology at Goenka Nursing Home, Guwahati. The investigation was conducted after receiving approval from the hospital's concerned authorities. The investigation was primarily concerned with the reproductive Assamese women who have PCOS and have visited the hospital for treatment for infertility and reproductive health issues. A total of 100 Assamese caste women within the age group between 18–42 years suffering from PCOS were selected. All the participants were enrolled after obtaining informed consent to participate in the investigation. The PCOS reproductive women were divided into two groups: obese PCOS women and non-obese PCOS women. The participants were interviewed separately to obtain the relevant data. The anthropometric variables of weight, height and blood pressure (BP) were obtained using standard procedures. The body mass index (BMI) and waist-hip ratio (WHR) were calculated. Weight in kilograms was recorded using a weighing machine, and height was measured in an upright position without shoes using an anthropometer to the nearest 0.5 cm. The BMI was calculated by dividing weight in kilograms by height in meters squared, i.e., $BMI = \text{weight (kg)} / \text{height (m}^2\text{)}$. The women with a $BMI < 23.00 \text{ kg/m}^2$ were considered of normal weight and, therefore, categorized into the non-obese

PCOS women group. Women with a BMI $\geq 23.00 - 27.50 \text{ kg/m}^2$ were overweight and with a BMI $> 27.50 \text{ kg/m}^2$ obese; thus, overweight and obese women were categorized into the obese PCOS women group based on the Asian criteria of the WHO [27]. The biological variables were compared between obese and non-obese PCOS women. The included variables were testosterone, fasting serum insulin, serum triglycerides (TG), total cholesterol (TL), fasting glucose, low density lipoprotein (LDL), high density lipoprotein (HDL), luteinizing hormone (LH), follicle stimulating hormone (FSH), LH:FSH, menstrual irregularity, and hypertension that were obtained from the test reports of the women concerned. Menstrual irregularity was calculated by considering 24–28 days as normal and > 38 days as oligomenorrhea.

Inclusion criteria: Women with PCOS-related infertility aged < 42 years were diagnosed with PCOS as per Rotterdam criteria. Women with oligoovulation and/or anovulation, hyper-androgenism, polycystic ovaries in the reproductive age group of 18–42 years who were willing to give informed consent were included in the present investigation.

Exclusion criteria: However, research participants who had consulted a psychiatrist or were already diagnosed with a mental illness could self-report on their history and peruse past medical records. Women on any insulin-sensitizing agent, lipid-lowering agent, or having any endocrine disorder were excluded from the study.

Statistical analysis

The collected data were entered into Microsoft Excel and analyzed in the Statistical Package for Social Sciences (Version 16.0). The continuous variables were depicted in terms of descriptive statistics of mean, standard deviation, and range distribution. The Chi-square test was performed to determine the frequency differences in categorical variables. The Mann-Whitney *U*-test was conducted for the statistical analysis of skewed continuous variables. The *p*-value of 0.05 was considered statistically significant.

RESULTS

Out of 100 Assamese women with PCOS, 71 women or 71.00% were overweight or obese, defined as having a BMI between 23.0–27.5 kg/m², whereas 29 women or 29.00% were non-obese, defined as having a BMI under 23.0 kg/m² (Table 1).

Table 1. BMI distribution of PCOS women based on Asian criteria of the WHO [27]

BMI category (kg/m ²)	Class of BMI	Distribution Mean ± SD, (No)
< 23.00	Underweight and normal weight	21.46 ± 0.93 (29)
23.00–27.50	Increased risk of metabolic syndrome	24.50 ± 0.77 (50)
≥ 27.50	High risk of metabolic syndrome	30.23 ± 0.22 (21)

The comparison of clinical, metabolic and hormonal parameters among the obese and non-obese PCOS groups is depicted in Table 2. Biochemical hyperandrogenism (testosterone) is higher in obese PCOS women than in non-obese PCOS women (69.22 ± 14.40 vs. 67.08 ± 12.69 ng/dL, $p > 0.05$), but the results are statistically insignificant. Fasting serum insulin was found to be significantly higher among obese PCOS women as compared to non-obese PCOS women (14.01 ± 5.50 vs. 10.45 ± 5.23 mIU/L, $p < 0.05$). High fasting glucose level was common and was found to be higher among obese PCOS women (92.28 ± 19.25 vs. 84.28 ± 15.56 mg/dL, $p < 0.05$), and the results were statistically significant. The metabolic parameters of serum triglycerides (TG), total cholesterol (TL), low density lipoprotein (LDL), and high density lipoprotein (HDL) were also found to be higher among obese PCOS women compared to non-obese PCOS women – TG: 161.30 ± 13.36 vs 137.03 ± 12.61 mg/dL, $p < 0.05$, TL: 180.10 ± 21.86 vs 171.21±24.81 mg/dL, $p < 0.05$, LDL: 120.21 ± 13.47 vs 109.45 ± 14.72 mg/dL, $p < 0.05$ and HDL: 55.44 ± 10.40 vs 51.55 ± 10.76 mg/dL, $p < 0.05$ respectively. The results for TG, TL, LDL and HDL were found to be statistically significant. The results showing the ratio of LH:FSH, which is one of the indicators of PCOS, showed a higher value among non-obese PCOS women in comparison to obese PCOS women (2.59 ± 0.36 vs. 2.13 ± 0.22 IU/L, $p < 0.05$), and the findings were statistically significant. Menstrual irregularity was statistically more common among obese PCOS women as compared to non-obese PCOS women and was found to be higher among obese PCOS women (87.32% vs. 65.52%, $p < 0.05$).

Table 2. Comparison of clinical, metabolic and hormonal parameters among the obese and non-obese PCOS groups

Parameters	Mean distribution (n = 100), (mean ± SD), (%)	Obese PCOS: BMI > 23 kg/m ² , (n = 71) (Mean ± SD), (%)	Non-Obese PCOS: BMI < 23 kg/m ² , (n = 29) (Mean ± SD), (%)	Mann Whitney U Test	
				z-score	p-value
Testosterone (ng/dL)	68.60 ± 13.96	69.22 ± 14.40	67.08 ± 12.69	0.46337	0.64552
Fasting serum insulin (mIU/L)	12.98 ± 5.66	14.01 ± 5.50	10.45 ± 5.23	3.03092	0.00244
Serum triglycerides (mg/dL)	154.26 ± 17.15	161.30 ± 13.36	137.03 ± 12.61	6.27834	< 0.00001
Total cholesterol (mg/dL)	177.52 ± 23.11	180.10 ± 21.86	171.21 ± 24.81	2.04720	0.04036
Fasting glucose (mg/dL)	89.96 ± 18.61	92.28 ± 19.25	84.28 ± 15.56	2.00542	0.04444
LDL (mg/dL)	117.38 ± 14.96	120.21 ± 13.47	109.45 ± 14.72	3.27780	0.00104
HDL (mg/dL)	54.31 ± 10.65	55.44 ± 10.40	51.55 ± 10.76	2.10038	0.03572
LH (IU/L)	14.37 ± 6.32	14.98 ± 6.38	12.90 ± 5.94	2.35105	0.01878
FSH (IU/L)	6.39 ± 3.40	6.54 ± 3.29	6.03 ± 3.64	1.29517	0.19360
LH:FSH	2.26 ± 0.34	2.13 ± 0.22	2.59 ± 0.36	-5.69342	< 0.00001
Menstrual irregularity (Yes)	81 (81)	62 (87.32)	19 (65.52)	Chi ² = 6.321	0.01166
Menstrual irregularity (No)	19 (19)	9 (12.68)	10 (34.48)		

The comparison of anthropometric parameters between obese PCOS and non-obese PCOS women is presented in Table 3. The results showed that the average age of obese PCOS and non-obese PCOS women was respectively 31.69 ± 4.81 years and 28.34 ± 6.63 years. The average weight (kg) was 62.38 ± 4.57 , which was higher in the obese PCOS women group than in the non-obese PCOS women group (51.23 ± 3.56). Height (cm) was found to be 155.97 ± 5.48 , which was higher in the obese PCOS group than in the non-obese PCOS group (153.62 ± 4.29). The variables of BMI, WC, HC and WHR were found to be higher among obese PCOS women as compared to non-obese PCOS women – BMI: 26.20 ± 2.70 vs. 21.46 ± 0.93 kg/m², $p < 0.05$), WC: 82.35 ± 5.22 vs. 78.12 ± 5.06 cm, $p < 0.05$, HC: 90.21 ± 5.34 vs. 84.11 ± 6.52 cm, $p < 0.05$, and WHR: 0.90 ± 0.32 vs. 0.82 ± 0.03 kg/m², $p < 0.01$ respectively. Blood pressure was also found to be higher in obese PCOS women than in non-obese PCOS women, with systolic blood pressure being 131.16 ± 10.36 vs. 124.21 ± 10.71 mm/Hg,

$p < 0.05$, and diastolic blood pressure was 84.72 ± 8.05 vs. 78.45 ± 7.16 mm/Hg, $p > 0.05$ respectively, and the results were statistically significant. The findings showed that weight, BMI, WC, HC, WHR, and blood pressure showed statistically significant results.

Table 3. Comparison of anthropometric parameters among the obese and non-obese PCOS women groups

Parameters	Obese PCOS: BMI > 23 kg/m ² , (n = 71), (Mean ± SD)	Non-Obese PCOS: BMI < 23 kg/m ² , (n = 29), (Mean ± SD)	Mann-Whitney U-test	
			z-score	p-value
Age (years)	31.69 ± 4.81	28.34 ± 6.63	2.35865	0.01828
Weight (kg)	62.38 ± 4.57	51.23 ± 3.56	7.40259	< 0.00001
Height (cm)	155.97 ± 5.48	153.62 ± 4.29	1.67878	0.09296
Body mass index (kg/m ²)	26.20 ± 2.70	21.46 ± 0.93	7.81659	< 0.00001
Waist circumference (cm)	82.35 ± 5.22	78.12 ± 5.06	3.13727	0.00168
Hip circumference (cm)	90.21 ± 5.34	84.11 ± 6.52	4.31849	< 0.00001
Waist/hip ratio (kg/m ²)	0.90 ± 0.32	0.82 ± 0.03	7.81659	< 0.00001
Systolic blood pressure (mm/Hg)	131.16 ± 10.36	124.21 ± 10.71	3.85892	0.00012
Diastolic blood pressure (mm/Hg)	84.72 ± 8.05	78.45 ± 7.16	3.43352	0.00060

Table 4 shows Spearman's rank correlation of BMI with anthropometric parameters among obese and non-obese PCOS women. The results revealed a positive correlation of WC with obese PCOS women ($r = 0.05776$, $p > 0.05$), while a negative correlation was found with non-obese PCOS women ($r = -0.1556$, $p > 0.05$). HC showed a negative correlation with obese PCOS women ($r = -0.49$, $p < 0.05$) and a positive correlation with non-obese PCOS women ($r = 0.4016$, $p > 0.05$). There was a negative correlation between WHR and obese PCOS women ($r = -0.4738$, $p < 0.05$) and a positive correlation between WHR and non-obese PCOS women ($r = 0.00925$, $p > 0.05$). Blood pressure levels also showed a negative correlation with obese PCOS women ($r = -0.4762$, $p < 0.05$ and $r = -0.4926$, $p < 0.05$) and a positive correlation with non-obese PCOS women ($r = 0.01482$, $p > 0.05$ and $r = 0.001235$, $p > 0.05$) respectively.

Table 4. Spearman's rank correlation of body mass index with anthropometric parameters among the obese and non-obese PCOS women groups

Parameters	Spearman rank & correlation test	Obese PCOS: BMI > 23 kg/m ² , (n = 71)	Non-obese PCOS: BMI < 23 kg/m ² , (n = 29)
Waist circumference (cm)	r-value	0.005776	-0.1556
	p-value	0.9619	0.4202
Hip circumference (cm)	r-value	-0.49	0.04016
	p-value	0.000014	0.8361
Waist/hip ratio	r-value	-0.4738	0.00925
	p-value	0.00003	0.962
Systolic blood pressure (mm/Hg)	r-value	-0.4762	0.01482
	p-value	0.000027	0.9392
Diastolic blood pressure (mm/Hg)	r-value	-0.4926	0.001235
	p-value	0.000013	0.9949

Table 5 compares the mean BMI levels of the PCOS women to observe the prevalence of obesity among Indian women and non-Indian women. The mean BMI level of the Indian women with PCOS was reported to range from 25.03 kg/m² to 33.70 kg/m² [26, 28–32]. Among non-Indian women with PCOS, the mean BMI ranges from 22.5 kg/m² to 33.28 kg/m² [2, 19, 33–36].

Table 5. Comparative table showing the prevalence of obesity in Indian and non-Indian women based on their mean BMI

Indian women with obesity and PCOS						
Population	Sample size	Study nature	Mean BMI level(kg/m ²)	Overweight/obesity	PCOS	References
North Indian women	450 (300 obese and 150 lean)	Prospective study	27.50	Obese = 66.6% (N = 300)	100.00%	Mazumdar et al. [28]
Women from Haryana	85 (PCOS) 85 (control)	Case-control study	25.03	41.17% overweight; 5.88% obese	50.00%	Shah et al. [29]
South Indian women	86 PCOS women	Observational case control study	27.93	11.62% Overweight; 69.76% Obese	100.00%	Kiranmayee et al. [30]
Eastern Indian women	58 PCOS women	Comparative study	28.14	Obese=65.51% (N=38)	100.00%	Jena et al. [26]
Bengali women	66 PCOS women	Cross-sectional study	29.84	27.30% Obese PCOS women	100.00%	Bhattacharya et al. [31]
Asian women	90 PCOS 90 control	Observational case-control study	30.06	18.88% Obese (n=17)	50.00%	Kumawat et al. [32]
Non-Indian women with obesity and PCOS						
British women	319 women with PCOS and 1060 age-matched control women	Retrospective cohort study	27.50	Obese PCOS 23.51% (N = 75)	23.13%	Wild et al. [2]
Korean women	837 PCOS women	Cross-sectional observational study	22.50	Obese PCOS = 11.90% (n = 268)	100.00%	Kim et al. [19]
Pakistani women	204 (50 controls, 65 PCOS, 25 first degree relatives)	Cross-sectional and analytical study	22.63	Obese PCOS = 81.54%	46.00%	Akram et al. [33]
Tehran women	754 (704 eumenorrheic non-hirsute subjects and 50 PCOS women)	Population-based study	28.69	Obese PCOS = 54.00%	7.10%	Behboudi-Gandevani et al. [34]
Istanbul women	88 (48 PCOS, 40 controls)	Prospective cohort study	33.28	Obese PCOS = 60.41% (N = 29)	54.54%	Nehir et al. [35]
Arabian women	63 PCOS women	Retrospective case-control study	30.30	Obese PCOS women = 52.0% (N = 33)	100.00%	Saadia et al. [36]

DISCUSSION

Overweight or obese women account for approximately 80% of PCOS patients, and there is an association between these conditions and PCOS [17, 37]. The present study found that 71.00% of PCOS women were overweight or obese (Table 1). Insulin resistance, which is common in the majority of obese PCOS patients, raises the risk of developing type-2 diabetes. Insulin levels in obese PCOS women are higher and of greater magnitude than in non-obese PCOS women (Table 2). Serum testosterone, fasting serum insulin, serum triglycerides, LDL, HDL, LH/FSH ratio, and menstrual irregularity were found to be statistically significant (Table 2). In line with the findings of Sachdeva et al. [1], the study discovered that obese PCOS women had higher testosterone levels than non-obese PCOS women. Faraji et al. [38] stated that more than 50% of PCOS patients were obese, and they observed that increased androgen and insulin resistance were the main etiologies of the disease. Obese women with PCOS have a more severe metabolic and hormonal profile than non-obese women with PCOS [1, 6, 20, 38]. These women have increased production of androgens in their ovaries and adrenal glands as a result of excessive hormone conversion in adipose tissue or as a result of hyperinsulinemia [20, 39]. The reduced concentration of sex-hormone-binding globulin may also cause the pool of free hormones to be higher than in non-obese women [21]. Due to these disorders, obese women remain at increased risk of developing menstrual disorders, hirsutism, and PCOS [6, 7, 10, 40, 41]. Obesity is a major contributor to insulin resistance, and secondary hyperinsulinism favours hyperandrogenism and PCOS [5–7, 42, 43]. Obesity plays a significant role in PCOS, and while it is not a diagnostic criterion, it appears to exacerbate many aspects of the PCOS phenotype [44]. However, compared to women without PCOS, obese and non-obese PCOS women have more visceral adipose tissue, and their levels of total androgen are positively correlated with each other [26, 42, 43, 45]. This indicates that obesity plays a significant role in PCOS. The presence of obesity may exacerbate reproductive and metabolic disorders in the form of insulin resistance, type 2 diabetes, and abnormalities in blood pressure regulation [9, 46, 47]. The findings showed that obese PCOS women exhibited significantly higher serum testosterone levels than non-obese PCOS women ($p < 0.05$, Table 2), which clearly indicates that androgens are closely related to obesity in PCOS women [10, 20, 21, 24, 39]. A study by Kim et al. [19] found a higher level of hyperandrogenism in women with a high BMI. Hormonal imbalances result from obesity, which increases hyperandrogenism and increases fasting serum insulin [1, 5, 7, 10, 42, 43, 47, 48]. In the present study, hyperandrogenism was

found to be present in both obese and non-obese PCOS women, but obesity was found to worsen the condition, which can be corroborated with the study of Nehir et al. [35]. Obesity increases the risk of insulin resistance and is commonly characterized by increased cholesterol levels and lipid accumulation, which lead to dysfunction in the ovaries [42, 43, 49, 50]. Obesity mainly manifests itself in increased levels of total cholesterol, triglycerides, and various apolipoprotein abnormalities [10, 30, 43, 51], which therefore promote insulin resistance in PCOS women [9, 23, 43, 46]. That could also be observed in the present investigation (Table 2). PCOS is considered to be a metabolic disorder as well as a reproductive disorder owing to its association with obesity and hyperandrogenism [35, 42, 43]. Increased LH and FSH levels in obese and non-obese women with PCOS were found in the present investigation, which is consistent with findings from earlier studies [5, 33, 36] and indicates that PCOS has reproductive disorder characteristics. Obesity was associated with suppressed ovulation and high LH levels, and the LH/FSH ratio was significantly increased in women with PCOS [5, 6, 21, 33, 36, 52, 53]. Obese PCOS women frequently have irregular menstrual cycles [36, 54, 55]. Menstrual irregularity in overweight and obese PCOS women was found to be 87.32% and 71.00% respectively (Table 2). According to Castillo-Martinez et al. [56], the prevalence of menstrual irregularity among obese PCOS women was 34.40%, which was lower than the prevalence discovered in the present investigation. Independent of type-2 diabetes, there was also a link between obesity and irregularities in the menstrual cycle [56]. In the present study, obese PCOS women had a higher prevalence of increased fasting glucose level, indicating a link between obesity and type-2 diabetes. This result prompts us to propose, similarly to other researchers [42, 43], that obesity, although it does not directly cause PCOS, may allow for its phenotypic expression in women who are predisposed to it by causing insulin resistance and hyperinsulinemia. Obesity is considered a key factor in the alteration of blood pressure in PCOS women. Higher cardiovascular risk was implicated in PCOS with aging and its consequent association with both systolic and diastolic blood pressure [57]. The present investigation showed a prevalence of 5.63% of hypertension disorders among obese PCOS women. The increased prevalence of hypertension in PCOS women has been linked to insulin resistance, hyperinsulinemia, hyperandrogenism, and obesity [6, 7, 58–60]. The present investigation highlighted the importance of anthropometric indices of obesity among PCOS women. In this study, BMI showed a significant association with two other anthropometric parameters (e.g., body weight and height) among the women, as it is a well-known derived parameter

for obesity [17, 31]. An increase in some anthropometric indices can be used to determine the prevalence of central obesity [29, 30, 32, 38]. In the vast majority of obese PCOS women, anthropometric parameters were above the threshold cut-off value (Table 3). The most common anthropometric abnormalities observed were weight, BMI, waist circumference, WHR, and systolic blood pressure among the obese PCOS women (Table 3). Kumawat et al. [32] have also found that anthropometric variables such as WC, HC, and WHR were significantly high in women with PCOS, which was quite relatable to the present study. The WHR seems to be an anthropometric index showing the highest predictive value for metabolic risk in PCOS-WHR, obese women [30, 32, 34]. Kiranmayee et al. [30] investigated the anthropometric parameters of PCOS women in order to study their lipid profiles and discovered abnormalities in waist circumference, WHR, and BMI. The prevalence of the metabolic syndrome increases with increasing BMI and WHR. WHR has been shown to be a reliable indicator of central obesity in PCOS women [17, 29, 31, 32, 55]. Table 4 shows a positive correlation between BMI and obese PCOS women. Thus, a high BMI would result in ovulation problems, leading to irregular menstrual cycles and PCOS in obese women. Table 5, which demonstrates the prevalence of obesity across the various demographics of India and non-Indian women with PCOS, provides more evidence that PCOS and obesity are strongly associated. According to Mazumdar et al. [28], prospective research was carried out among North Indian women where it was discovered that the mean BMI level of the PCOS women was 27.50 kg/m² and that 66.66% of the 450 PCOS women were obese. According to a case-controlled study, 85 out of 170 women in Haryana or 50.00% had PCOS, and their mean BMI was 25.30 kg/m², with obesity at 5.88% and overweight at 41.17% [29]. According to Kiranmayee et al. [30], who studied 86 PCOS women, the prevalence of overweight and obese PCOS women in South India was 11.62% and 69.76%, respectively, with a mean BMI of 27.93 kg/m². Additionally, studies by Jena et al. [26], Bhattacharya et al. [31], and Kumayat et al. [32] found that the mean BMI level was 28.14 kg/m², with obesity found to be 65.51% among 58 PCOS women, 29.84 kg/m² with obesity found to be 27.30% among 66 PCOS women, and 30.06 kg/m² with obesity of 18.88% among 90 PCOS women. These studies were carried out on women from Eastern India, Bengal, and Asia, respectively. In contrast to studies on Indian women, few studies have examined the prevalence of obesity and PCOS in non-Indian women, Table 5 indicates that Wild et al. [2] conducted a retrospective cohort study among British women with a sample size of 1379, 319 of whom had PCOS and a mean BMI of 27.50 kg/m²,

with obesity found to be 23.51% ($N = 75$) and PCOS prevalence found to be 23.13%. According to Kim et al. [19], the obesity rate among the 837 Korean PCOS women was 11.90%, with the mean BMI level being 22.50 kg/m². According to Akram et al. [33], 204 Pakistani women had an average BMI of 22.63 kg/m²; 65 of them or 46.00% had PCOS, and 81.54% of the PCOS women were obese. Similarly, studies by Behboudi-Gandevani et al. [34]; Nehir et al. [35], and Saadia et al. [36] reported a mean BMI level of 28.69 kg/m² with a 54.00% obesity level, and PCOS prevalence was found to be 7.10% in a total sample size of 754 women, out of which 50 were PCOS cases and the rest were eumenorrheic non-hirsute subjects; a mean BMI level of 33.28 kg/m² had an obesity prevalence of 60.41% among 48 PCOS cases (54.54%); 30.30 kg/m² had an obesity of 52.00% among 63 PCOS women, and the subjects of these investigations were women from Tehran, Istanbul, and Arabia, respectively. As a result, PCOS was revealed to be a clinically diverse illness with a higher incidence of obesity.

STUDY LIMITATIONS

First, the present investigation primarily focused on a specific community inside a specific area, and the time spent with the women was brief. It was also based on a limited research participants visiting the gynecology department, which may have an impact on generalizing the study findings.

CONCLUSION

Obese women with PCOS are more likely to experience poor metabolic and reproductive results. The anthropometric measurements of PCOS women were discovered to be closely related to obesity. However, the present study is based on a small sample size; therefore, further study is recommended with a large sample size focusing on ethnicity in Northeast India.

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Address for Correspondence:

Dr. Chumi Das

Department of Anthropology, Cotton University

Panbazar, Guwahati 781001, Assam, Northeast India

E-mail: chumianth@gmail.com