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Original Research

Body Fat Distribution in Thai Reproductive-Aged Polycystic Ovary Syndrome Women Compared with Non-Polycystic Ovary Syndrome Women

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Abstract

Background: The body fat in polycystic ovary syndrome (PCOS) women is mostly centrally distributed and is associated with insulin resistance, diabetes mellitus, and hyperandrogenemia. This study compared the fat distributions of Thai PCOS and non-PCOS women, and it investigated the association between body fat distribution in PCOS women with glucose tolerance and serum androgens. **Methods**: The PCOS and non-PCOS groups each had 60 women. The body mass indexes (BMI) of the groups were matched. Blood tests and fat distributions were compared between group. **Results**: The mean age of the non-PCOS group was significantly higher than that of the PCOS group (30.85 $\pm$ 6.41 *vs.* 25.95 $\pm$ 5.16 years; p-value $<$ 0.001). The glucose level after a 2-hour, 75-gram, oral glucose tolerance test (75-g OGTT) of the PCOS group, and its insulin resistance, triglyceride, low-density lipoprotein, total testosterone, free testosterone, and dehydroepiandrosterone sulphate levels, were significantly higher than the corresponding values of the non-PCOS group. The fat distribution patterns of the 2 groups were generally not significantly different. The level of fat distributed in the arms was significantly elevated among PCOS women with abnormal 75-g OGTT values. The fat distributions of PCOS women, regardless of hyperandrogenemia status, did not significantly differ. **Conclusions**: No significant differences in fat distribution were observed between the PCOS and non-PCOS groups. PCOS participants with abnormal 75-g OGTT levels had a higher proportion of arm-fat compared to those with normal results. There were no discernible differences in fat distribution patterns between PCOS women with hyperandrogenemia and those with normal androgen levels.

Keywords: fat distribution; glucose intolerance; polycystic ovary syndrome; hyperandrogenemia

1. Introduction

Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder that impacts reproductive-aged women, with an prevalence ranging from 4% to 7% [1]. The 2003 Rotterdam criteria are the most widely accepted diagnostic guidelines, requiring the presence of a minimum of two of the following criteria [2]: (1) oligo- or anovulation; (2) clinical and/or biochemical indications of hyperandrogenism; and (3) the presence of polycystic ovaries or polycystic ovarian morphology. Additionally, it is imperative to rule out other endocrinopathies that may share a similar clinical presentation or mimic PCOS.

The development of PCOS has been attributed to pathological changes in the endocrine and metabolic systems, resulting in obesity, glucose intolerance, metabolic syndrome (MS), and diabetes mellitus [1,3–5]. A prior investigation conducted at the Department of Obstetrics and Gynecology, Faculty of Medicine Siriraj Hospital, reported a 20% prevalence of abnormal glucose tolerance test results among PCOS patients [6]. Moreover, this investigation indicated that the prevalence of MS in PCOS was 18.0%–21.2% by difference criteria [7]. Recent evidence has indicated that the fat distribution pattern is a risk factor for various metabolic disorders [1,5,8,9]. PCOS women typically exhibit central or abdominal fat accumulation, characterized as android fat distribution [5,10]. A study revealed that Thai PCOS women exhibited an elevated body mass index (BMI), greater central obesity, and a higher visceral adiposity index (VAI) compared to normal women [11]. This particular at distribution pattern is associated with glucose intolerance, hyperinsulinemia, diabetes mellitus, and hyperandrogenemia [12,13].

Bioelectric impedance analysis (BIA) is a currently widely used method for assessing body fat distribution [14]. BIA stands out due to its simplicity, noninvasiveness, cost-effectiveness, and portability, making it suitable for a broad

range of subjects. Moreover, as it uses a bioelectrical impedance method, avoiding any exposure to radiation [14–18]. Furthermore, research has shown that the accuracy of BIA is comparable with that of the gold standard method [16,19,20].

BIA is a method for assessing body fat composition by measuring electrical impedance, the resistance to low-amperage electric current (<1 ampere) as it travels through body tissues [17,21,22]. The analysis involves a user standing on four metal electrodes and holding four hand-held electrodes. A minimal, safe, and painless electrical signal originating at each foot passes through the legs, buttocks, trunk, and arms [14,17,19]. The outcomes provide precise accurate measurements of the degrees of body fat, muscle mass, and water content in each body segment [19].

In earlier studies [9,10,12,13,23], analyses involved comparisons between PCOS patients and diverse control cohorts to elucidate patterns of body fat distribution. Unfortunately, a dearth of data exists concerning Asian women with PCOS. Hence, the present work set out to compare the body fat distributions of PCOS and non-PCOS women. A secondary objective was to determine whether body fat distribution is associated with glucose intolerance and serum androgens in Thai PCOS women.

2. Materials and Methods

A cross-sectional study was conducted at the Gynecologic Endocrinology Unit, Department of Obstetrics and Gynecology, Faculty of Medicine Siriraj Hospital. Subjects were enrolled between May 2020 and November 2020. Before commencement of this research, its protocol received approval from the Siriraj Institutional Review Board (Si 180/2020).

The sample size was determined based on the findings of a pilot study, which reported mean fat mass values at the trunk region of 13.47 $\pm$ 6.00 kilograms (kg) for PCOS women and 15.82 $\pm$ 5.22 kg for non-PCOS (control) women. In the current investigation, it was decided to match the sample sizes and the BMIs of a PCOS and control group, using a paired sample *t*-test. A minimum required sample size of 54 was calculated for each group. After adjusting for incomplete data of 10%, the total sample size came to 120 (60 per group).

Sixty PCOS women, diagnosed according to the 2003 Rotterdam criteria, were included in the study group. All were aged between 18 and 45 years. A further 60 non-PCOS women were recruited for the control group. Non-PCOS were recruited from volunteers who expressed interest in engaging with the research project. The non-PCOS cohort, who aged 18 to 45 with regular menstrual cycles and no underlying medical conditions, was identified through a comprehensive evaluation, including a detailed history-taking focused on menstrual regularity, the absence of hyperandrogenism manifestations, and confirmation through pelvic ultrasonography revealing normal ovarian morphology. Both groups were matched by BMI. Individuals were excluded from either group if they had utilized hormonal contraception within the preceding 3 months or any medication capable of influencing insulin sensitivity, fat deposition, or muscle mass within the preceding 6 months. Additionally, participants with contraindications for BIA, a history of fat distribution-related surgeries (e.g., liposuction), pregnancy, or lactation were ineligible for enrollment.

Medical histories of the PCOS and control group were obtained, followed by comprehensive physical examinations encompassing height, weight, waist circumference, and blood pressure assessments. The degree of hyperandrogenism was documented, with acne classified as mild, moderate, or severe [24], alopecia graded using the Ludwig classification system [25], and hirsutism evaluated through the modified Ferriman-Gallwey score (mFGS) [26]. This study adopted the mFGS >4 as the cut-off value of hirsutism, based on a study from China [27]. BMI was categorized in accordance with World Health Organization (WHO) Asian-BMI criteria [28]: underweight (BMI <18.5 kg/m^2); normal (BMI 18.5–22.9 kg/m^2); overweight (BMI 23.0–24.9 kg/m^2); obese level I (BMI 25.0–29.9 kg/m^2); and obese level II (BMI <30.0 kg/m^2). Urine pregnancy tests were performed to exclude pregnancy.

Venous blood samples were obtained on two occasions. The initial sample was obtained following an overnight fast of a minimum of 8 hours, typically 8:00–10:00 AM. This initial sample included assessments of fasting plasma glucose, insulin, dehydroepiandrosterone sulphate (DHEA-S), sex hormone-binding globulin (SHBG), testosterone, albumin, and lipids (triglyceride, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and cholesterol). A subsequent blood test, measuring serum glucose levels 2 hours post a 75-gram oral glucose challenge (2- hour 75-g glucose), was conducted. Free testosterone level was determined using an online calculator created by the International Society for the Study of the Aging Male (ISSAM) [29]. The laboratory assays were conducted by the Department of Clinical Pathology, Faculty of Medicine Siriraj Hospital, Mahidol University, using an automated analyzer (Cobas 8000; Roche Diagnostics, Rotkreuz, Switzerland). The blood tests for cholesterol, triglyceride, LDL-C, HDL-C, and albumin were analyzed by the enzymatic colorimetric method. Electrochemiluminescence immunoassay was used to measure SHBG, DHEA-S, testosterone, and insulin levels. Glucose levels was determined enzymatically by the hexokinase method. The intra- and inter-assay coefficients of variation were below 5% for all methods.

Following the initial venous blood sample collection, participants' body fat distributions were assessed utilizing a certified BIA device (Tanita Health Equipment HK Ltd., Hong Kong, China), which undergoes annual local quality control checks and holds ISO 9001 certification. Fat levels were evaluated in both the upper (both arms and the trunk)

and lower body regions (the buttocks and both legs). This assessment typically lasted approximately 1 minute, after which participants underwent the collection of the second venous blood sample.

In this study, abnormal 75-gram oral glucose tolerance test (75-g OGTT) values were classified in accordance with the 2016 American Diabetes Association criteria [30]. The categories included "impaired fasting glucose" (fasting glucose $>$100 and $<$126 mg/dL), "impaired glucose tolerance test" (2-hour plasma glucose $>$140 and $<$200 mg/dL), and "diabetes mellitus" (fasting glucose $>$126 mg/dL or 2-hour plasma glucose $>$200 mg/dL). Hyperandrogenemia was indicated by one of the following: (1) total testosterone $>$0.48 ng/mL, as per the International PCOS Network guidelines of 2018 [31]; (2) free testosterone $>$2%, determined using the online calculator of the ISSAM [29]; and (3) DHEA-S $>$350 ug/mL [32].

For statistical analysis, descriptive data were presented as means $\pm$ standard deviations, while continuous data were reported as medians with 25th and 75th percentiles (P25 and P75). In the case of categorical data, their percentages were calculated. The Chi-Square test, paired *t*-test, and Mann-Whitney U test were used for comparisons between the groups. Age adjustments were performed through analysis of covariance (ANCOVA). A p-value $<$ 0.05 was considered statistically significant. All statistical analyses were conducted using PASW Statistics for Windows (version 18.0; SPSS Inc., Chicago, IL, USA).

3. Results

Sixty women were in the PCOS group and additional sixty were in the control group. Among the PCOS women, 34 (57%) met all 3 Rotterdam criteria, while the remaining 26 (43%) met various combinations of two criteria. Specifically, fifteen PCOS cases (25%) were diagnosed based on oligo- or anovulation in conjunction with clinical and/or biochemical signs of hyperandrogenism, while an additional 9 (15%) met the criteria through oligo- or anovulation alongside polycystic ovarian morphology. The remaining two PCOS cases (3%) were diagnosed based on biochemical and/or clinical signs of hyperandrogenism along with polycystic ovarian morphology.

The characteristics as well as the biochemical and serum hormone measurements of the PCOS and control groups are detailed in Table 1. The control group exhibited a greater average age compared to the PCOS group (30.85 $\pm$ 6.41 years, *vs*. 25.95 $\pm$ 5.16 years; p-value $<$ 0.001). A majority of the participants in both groups were either overweight or obese. The values for the fasting insulin, 2-hour 75-g OGTT, and homeostatic model assessment of insulin resistance (HOMA-IR) were significantly greater for the PCOS group. Furthermore, lipid profile of the PCOS group was significantly different from that of the control group, characterized by elevated triglyceride and LDL-C levels. Similarly, androgen levels in the PCOS group (DHEA-S, free testosterone, and total testosterones) were significantly higher.

Table 2 provides a comparison of fat distributions between the PCOS and control groups. After age adjustment via ANCOVA, the distributions did not show a statistical a difference.

The fat distributions of the PCOS women with normal and abnormal 75-g OGTT results are compared in Table 3. The percentage of fat distributed in the arms was significantly higher for the PCOS women with abnormal 75-g OGTT values (p-value = 0.003).

Table 4 compares the fat distributions of the PCOS women with normal androgen levels and hyperandrogenemia. There were no significant differences in their distribution patterns.

Regarding the fat distribution patterns of the control group, there were no significant differences in the distributions for the women with normal and abnormal 75-g OGTT results (Table 5). Conversely, a statistically higher percentage of fat in the arms was noted in control women with hyperandrogenemia compared to those with normal androgen levels (p-value = 0.037; Table 6).

4. Discussion

The central, or android, fat distribution of PCOS women is associated with heightened risks for insulin resistance, diabetes, cardiovascular disease, and metabolic syndrome. Central obesity results from the accumulation of fat in subcutaneous and internal adipose tissue (the latter comprised of intra-abdominal and visceral adipose tissue). Excessive visceral fat accumulation is associated with insulin resistance. The mechanisms involved in the etiopathogenesis of such insulin resistance relate to pre-receptor, receptor, and post-receptor insulin defects. Visceral fat secretes adipokines that impair the insulin responsiveness of tissues such as the muscles and liver. This is characterized by decreased insulin access to muscle secondary to excessive free fatty acid (pre-receptor); insulin-receptor downregulation caused by hyperinsulinemia (receptor); and intracellular signaling pathway inhibition by various factors related to adiposity [33,34]. Numerous studies [1,7,8,23] have reported greater upper body fat distribution in PCOS patients compared to control groups of women. An earlier investigation at Siriraj Hospital found central obesity in 57% of overweight PCOS women and 49% of obese PCOS women [35]. Another study on Thai PCOS women revealed that central obesity presents a relative risk of 3.53 of developing glucose intolerance (95% confidence interval, 1.28–9.75) [6].

In an analysis of the body fat patterns among Caucasian PCOS women, Kirchengast and Huberet [36] found an extremely high prevalence of central fat distribution in PCOS women. Similarly, Douchi *et al*. [10] reported that fat was significantly prevalent in the upper part of the body

Table 1. Characteristics of the participants.

Characteristics	Control (n = 60)	PCOS (n = 60)	*p*-value
Age (years)	30.85 ± 6.41	25.95 ± 5.16	<0.001
BMI (kg/m^2)	24.02 ± 4.90	24.06 ± 4.84	0.997
Underweight	6 (10.00)	6 (10.00)	
Normal	21 (35.00)	22 (36.70)	
Overweight	10 (16.70)	9 (15.00)	
Obesity I	14 (23.30)	13 (21.70)	
Obesity II	9 (15.00)	10 (16.70)	
Waist circumference (cm)	79.32 ± 11.22	80.02 ± 10.64	0.727
Hip circumference (cm)	98.25 ± 10.69	97.67 ± 10.47	0.763
Waist-to-hip ratio	0.80 ± 0.05	0.81 ± 0.05	0.276
Fasting blood glucose (mg/dL)	80.70 ± 7.06	83.95 ± 11.51	0.065
2-hour 75-g OGTT (mg/dL)	96.47 ± 21.28	107.47 ± 33.45	0.034
Fasting insulin level (mIU/mL)	8.49 (6.12, 11.15)	11.18 (7.16, 16.65)	0.004
HOMA-IR	1.65 ± 1.82	2.30 ± 3.81	0.004
Cholesterol (mg/dL)	186.73 ± 35.75	190.25 ± 35.06	0.587
Triglyceride (mg/dL)	61.50 (46.00, 83.00)	75.50 (58.50, 96.50)	0.014
HDL (mg/dL)	67.48 ± 14.16	61.03 ± 15.34	0.018
LDL (mg/dL)	103.67 ± 29.74	116.16 ± 30.89	0.026
SHBG (nmol/L)	55.90 (38.85, 81.20)	36.10 (25.88, 60.53)	<0.001
Albumin (g/dL)	4.40 ± 0.21	4.48 ± 0.23	0.073
Total testosterone (ng/mL)	0.300 ± 0.140	0.490 ± 0.190	<0.001
Free testosterone (%)	1.31 ± 0.45	1.73 ± 0.60	<0.001
DHEAS (microgram/dL)	205.14 ± 85.62	250.56 ± 96.22	0.007

Data are presented as mean ± standard deviation (SD); median (P25, P75); or n (%). "BMI–Underweight" means <18.5 kg/m^2; "BMI–Normal" means 18.5–22.9 kg/m^2; "BMI-Overweight" means 23.0–24.9 kg/m^2; "BMI-Obesity I" means 25.0–29.9 kg/m^2; "BMI-Obesity II" means >30 kg/m^2. Abbreviations: 75-g OGTT, 75-gram oral glucose tolerance test; BMI, body mass index; DHEAS, dehydroepiandrosterone sulphate; HDL, high density lipoprotein; HOMA-IR, homeostatic model assessment of insulin resistance; LDL, low density lipoprotein; PCOS, polycystic ovary syndrome; SHBG, sex hormone-binding globulin.

of Asian PCOS women relative to healthy controls. However, the work of Jin *et al*. [12] revealed that Korean PCOS women had the same body fat distribution as the women in a control group. This corresponds with the findings of the current research, in which BIA did not identify any significant differences in the fat distribution of PCOS and control-group women.

BIA offers several advantages, including noninvasiveness, ease of use, rapidity, and the absence of radiation exposure [19]. BIA is commonly used for body composition assessments in both clinical settings and research investigations. The previously mentioned investigations evaluated fat distribution using methods other than BIA, such as computed tomography scanning and dual energy X-ray absorptiometry. Firm, overall conclusions about fat distribution might not be achievable when studies have used different evaluation methods.

The present work found that the mean age of the control group was significantly higher than that of the PCOS group. JafariNasabian *et al*. [37] proposed that aging is associated with changes in body composition and affects almost all physiological processes, with important consequences on health and physical functionality. Several studies have reported the amounts by which body fat mass increases throughout the lifespan [38–40]. As age may have affected the fat distribution findings of our study, the data were adjusted for age by ANCOVA. The results are listed in Table 2. The fat distributions of the non-PCOS and PCOS groups were not significantly different when adjusted for age.

Women with upper-body obesity have also been observed to exhibit reduced insulin sensitivity and an elevated risk of diabetes and cardiovascular disease [41]. Regardless of their BMI, women with PCOS have been found to display a substantial prevalence of upper-body obesity, as demonstrated by greater waist-hip ratio and waist circumference, relative to BMI-matched non-PCOS women [10,42]. Our study found a similar result to other studies in that the PCOS women with abnormal 75-g OGTT values had a significantly higher fat distribution in the arms than normal 75-g OGTT PCOS women. A similar result was observed in a study by Aydogdu *et al*. [43], who reported that fat accumulation in the arms was significantly higher in Caucasian PCOS women than healthy controls. However, the total

Table 2. Comparison of the fat distributions of the control and PCOS groups.

Body compartment	Fat mass distribution (kg)			Fat mass distribution (%)		
	Control (n = 60)	PCOS (n = 60)	*p-value	Control (n = 60)	PCOS (n = 60)	*p-value
Total body	20.53 ± 1.24	22.70 ± 1.24	0.236			
Upper part	13.12 ± 0.89	14.71 ± 0.89	0.224	62.06 ± 0.60	63.72 ± 0.60	0.062
-Arms	1.69 ± 0.13	1.96 ± 0.13	0.170	7.95 ± 0.14	8.23 ± 0.14	0.175
-Trunk	11.42 ± 0.76	12.75 ± 0.76	0.237	54.11 ± 0.55	55.50 ± 0.55	0.092
Lower part	7.41 ± 0.36	7.99 ± 0.36	0.278	37.94 ± 0.60	36.28 ± 0.60	0.062

*Adjusted for age by analysis of covariance (ANCOVA).

Data are presented as mean ± standard error (SE).

"Upper part" means fat in the arms and trunk.

"Lower part" means fat in the legs and buttocks.

Table 3. Comparison of the fat distributions of the PCOS women with normal and abnormal 75-gram oral glucose tolerance test (75-g OGTT) results.

Body compartment	Fat mass distribution (kg)			Fat mass distribution (%)		
	Normal	Abnormal	p-value	Normal	Abnormal	p-value
	75-g OGTT (n = 52)	75-g OGTT (n = 8)		75-g OGTT (n = 52)	75-g OGTT (n = 8)	
Total body	20.46 ± 9.09	29.98 ± 7.88	0.007			
Upper part	13.16 ± 6.43	19.76 ± 5.37	0.008	63.04 ± 3.99	65.60 ± 2.29	0.083
-Arms	1.70 ± 0.97	2.83 ± 0.99	0.004	7.95 ± 1.05	9.18 ± 1.09	0.003
-Trunk	11.46 ± 5.48	16.94 ± 4.41	0.009	55.09 ± 3.66	56.42 ± 1.99	0.319
Lower part	7.29 ± 2.69	10.21 ± 2.56	0.006	36.96 ± 3.99	34.39 ± 2.29	0.083

Data are presented as mean ± SD.

"Upper part" means fat in the arms and trunk.

"Lower part" means fat in the legs and buttocks.

Table 4. Comparison of the fat distributions of the PCOS women with normal androgen levels and hyperandrogenemia.

Body compartment	Fat mass distribution (kg)			Fat mass distribution (%)		
	Normal androgen level (n = 19)	Hyperandrogenemia (n = 41)	p-value	Normal androgen level (n = 19)	Hyperandrogenemia (n = 41)	p-value
Total body	20.89 ± 10.30	22.12 ± 9.14	0.645			
Upper part	13.50 ± 7.20	14.29 ± 6.46	0.672	63.40 ± 3.31	63.38 ± 4.18	0.981
-Arms	1.75 ± 1.15	1.90 ± 0.99	0.613	7.84 ± 1.32	8.25 ± 1.02	0.195
-Trunk	11.75 ± 6.07	12.39 ± 5.49	0.685	55.56 ± 2.68	55.13 ± 3.84	0.658
Lower part	7.39 ± 3.13	7.82 ± 2.72	0.585	36.59 ± 3.31	36.62 ± 4.18	0.981

Note: Data are presented as mean ± SD.

"Upper part" means fat in the arms and trunk.

"Lower part" means fat in the legs and buttocks.

body fat mass and trunk fat mass did not differ between the two study groups in the work by Aydogdu *et al*. [43]. Based on the aforementioned results, reducing the proportion of fat in the arms of PCOS women with abnormal OGTT values might reduce their risk for glucose intolerance. Among the control women in the current research, four out of the sixty participants had abnormal 75-g OGTT results. As to fat distribution, our study also compared the distributions of the non-PCOS women who had normal and abnormal 75-g OGTT values. There were no significant differences in their body fat distributions. These findings therefore raise doubts about a relationship between fat distribution and abnormal OGTT results for Thai PCOS women, which may be influenced by other factors. These factors could include the type of fat distribution across different body regions, which varies among individuals of different ethnicities. Additionally, there may be other mechanisms related to insulin resistance beyond fat distribution that contribute to the development of insulin resistance.

Most of the previous studies indicates that androgens exert a significant influence on the determination of body composition [1,9,41,44]. The predominant factor accounting for the upper-body fat distribution observed in women with PCOS is primarily attributed to excessive an-

Table 5. Comparison of the fat distributions of the non-PCOS women with normal and abnormal 75-gram oral glucose tolerance test (75-g OGTT) results.

Body compartment	Fat mass distribution (kg)			Fat mass distribution (%)		
	Normal	Abnormal	*p*-value	Normal	Abnormal	p-value
	75-g OGTT (n = 56)	75-g OGTT (n = 4)		75-g OGTT (n = 56)	75-g OGTT (n = 4)	
Total body	20.95 ± 9.41	29.13 ± 6.97	0.095			
Upper part	13.39 ± 6.79	19.33 ± 4.94	0.093	62.13 ± 5.09	66.16 ± 1.25	0.122
-Arms	1.74 ± 0.95	2.65 ± 0.99	0.069	7.99 ± 0.94	8.89 ± 1.22	0.079
-Trunk	11.65 ± 5.87	16.68 ± 3.95	0.099	54.13 ± 4.77	57.28 ± 0.81	0.196
Lower part	7.56 ± 2.69	9.80 ± 2.04	0.111	37.87 ± 5.09	33.84 ± 1.25	0.122

Note: Data are presented as mean ± SD.

"Upper part" means fat in the arms and trunk.

"Lower part" means fat in the legs and buttocks.

Table 6. Comparison of the fat distributions of the non-PCOS women with normal androgen levels and hyperandrogenemia.

Body compartment	Fat mass distribution (kg)			Fat mass distribution (%)		
	Normal androgen level (n = 45)	Hyperandrogenemia (n = 15)	*p*-value	Normal androgen level (n = 45)	Hyperandrogenemia (n = 15)	*p*-value
Total body	19.67 ± 8.16	26.98 ± 11.11	0.008			
Upper part	12.45 ± 5.78	17.81 ± 8.23	0.007	61.72 ± 5.02	64.45 ± 4.61	0.068
-Arms	1.61 ± 0.81	2.37 ± 1.19	0.032	7.91 ± 0.89	8.51 ± 1.09	0.037
-Trunk	10.84 ± 4.99	15.43 ± 7.09	0.008	53.81 ± 4.69	55.94 ± 4.37	0.128
Lower part	7.22 ± 2.46	9.17 ± 2.97	0.014	38.28 ± 5.02	35.55 ± 4.61	0.068

Note: Data are presented as mean ± SD.

"Upper part" means fat in the arms and trunk.

"Lower part" means fat in the legs and buttocks.

drogen levels [10,42,45]. Hyperandrogenism stands as a primary characteristic of PCOS, as heightened androgen levels represent its most consistent hallmark. In support of this pathophysiology, our study found that DHEA-S, free testosterone, and total testosterone levels were significantly higher for the PCOS than the non-PCOS women. Furthermore, excess androgen is associated with central fat distribution [1,43,44,46]. However, the present study did not identify any differences in the fat distribution patterns of the PCOS women with and without hyperandrogenemia. This finding also raises doubts about a relationship between central fat distribution and androgen levels for Thai PCOS women. While about 33% of the control group in our work had hyperandrogenemia, the women concerned had regular menstrual cycles and no clinical signs of the condition. This study revealed a higher fat distribution in the arms of the women in the control group who had hyperandrogenemia than for the control women with normal androgen levels. Upon closer examination, it becomes apparent that the trend among women with hyperandrogenism indicates a greater total body fat mass distribution than the group with normal androgen levels. This result may be explained by the fact that individuals with a higher fat mass may experience elevated androgen levels due to increased insulin resistance, compensatory hyperinsulinemia, and subsequent augmented androgen secretion by both the adrenal glands and the ovaries [47]. The involvement of adipose tissue in the development of PCOS is characterized by an elevated risk of obesity in affected women, particularly with a distinctive abdominal adiposity profile. Subsequently, this abdominal adiposity, which engages in mechanisms leading to insulin resistance, constituting a pivotal pathophysiological aspect of PCOS. This, in turn, initiates a vicious cycle. Additionally, adipose tissue has a profound effect on triggering excessive androgen production, a phenomenon associated with elevated insulin resistance. This is further compounded by compensatory hyperinsulinemia, leading to an augmented release of androgens from both the adrenal glands and the ovaries. These intricate interactions contribute to the exacerbation of symptoms in individuals with PCOS.

The anthropometric differences between Asians and Caucasians might include the former being thinner and having less central fat accumulation. These might be the effects of genetic, environmental, and/or lifestyle differences. Anthropometric differences might also be associated with the pathophysiology of PCOS. Many studies on the metabolic profiles of Caucasian PCOS women have reported that their profiles are worse than those of Asian PCOS women [9,13,36].

This study sheds light on the correlation between fat distribution and the development of insulin resistance. The

findings suggest that reducing the accumulation of fat in specific anatomical regions may have beneficial effects on insulin resistance. For example, engaging in targeted exercise focusing on those specific areas could potentially yield positive outcomes in terms of mitigating insulin resistance.

The strength of this study is that it successfully used a BIA device to compare the body fat distributions of reproductive-aged Thai women with and without PCOS. However, its weakness was its reliance on data collected from a sole institution's sample. Therefore, its findings might not represent all reproductive PCOS women in Thailand. For implication, if fat distribution in each body part effect to difference metabolic and androgenetic profiles, so controlling fat in effecting part could provide good metabolic and androgen profile.

5. Conclusions

There were no significant differences in the fat distributions of the PCOS and non-PCOS women. The PCOS women who had abnormal 75-g OGTT values had a higher percentage of fat in the arms than the PCOS women with normal 75-g OGTT values. The PCOS women who presented with hyperandrogenemia did not demonstrate a different fat distribution pattern than those with normal androgen levels.

Availability of Data and Materials

The data sets generated and analyzed during the current study are not publicly available due to confidentiality agreements, but are available from the corresponding author on reasonable request.

Author Contributions

Conceptualization: TW, PP; Project development: TW, PP; Methodology: TW, AK; Data Collection: PC, AK, SI, PT, MR, KT, SA, NS, NP; Statistical analysis: TW, PC, AK; Funding acquisition: TW; Manuscript writing: TW, AK; Manuscript editing: TW, PC. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript.

Ethics Approval and Consent to Participate

The study's protocol received approval from the Siriraj Institutional Review Board (Si 180/2020). All participants received comprehensive information regarding the study and furnished written consent prior to their participation.

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Conflict of Interest

The authors declare no conflict of interest.

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