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A COMPREHENSIVE ANALYSIS OF BODY COMPOSITION, METABOLIC DYSFUNCTION, AND HORMONAL PATTERNS IN PCOS WOMEN

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ABSTRACT **Objective:** To delineate the impact of body fat on glucose metabolism and hormonal profiles in women with polycystic ovary syndrome (PCOS) compared to healthy controls. **Methods:** A cross-sectional analysis was conducted on 133 PCOS patients and 140 age-matched controls at a tertiary care centre in southern India. Body composition was assessed via bioelectric impedance, while insulin resistance was evaluated using HOMA-IR and related indices. Hormonal levels (FSH, LH, E2, PRL, testosterone, and androstenedione) were measured biochemically. Participants were stratified by waist-to-hip ratio (WHR: central obesity ≥0.85) and body fat percentage (BFP: obese ≥35%). Statistical analyses included t-tests, Wilcoxon rank-sum tests, and linear regression. **Results:** Women with PCOS exhibited significantly worse insulin resistance (HOMA-IR) than controls, regardless of obesity status (central obesity P=0.011; non-central obesity P<0.001; high BFP P<0.001; low BFP P<0.001). Each incremental rise in BFP worsened insulin sensitivity more severely in PCOS patients. Hormonal imbalances (elevated LH, LH/FSH ratio, testosterone, and androstenedione) persisted across all PCOS subgroups. Notably, body fat indices (BFP, BMI) negatively correlated with LH and androstenedione in PCOS women but not in controls. **Conclusion:** Both obese and non-obese PCOS patients demonstrate greater metabolic and hormonal dysfunction than healthy women. Body fat exacerbates sex-hormone disturbances exclusively in PCOS, suggesting that clinical guidelines should incorporate body composition-specific management strategies.

KEYWORDS : Polycystic Ovary Syndrome, Body Fat Percentage, Insulin Resistance

INTRODUCTION

Polycystic ovary syndrome (PCOS), a perplexing and heterogeneous endocrinopathy, affecting 5%–20% of reproductive-aged women, presenting a labyrinth of gynecological, hyperandrogenic, and metabolic maladies—obesity, dyslipidemia, hyperinsulinemia, and insulin resistance (IR)—yet their intricate interplay remains enigmatic (1,2). While obesity, particularly abdominal adiposity, amplifies metabolic mayhem in half of PCOS patients, even lean women (40%–50% of cases) face heightened insulin insensitivity and fat distribution aberrations (3,4). Body fat percentage (BFP) and visceral fat, not merely BMI, dictate metabolic risks, with central obesity emerging as a sinister sentinel of cardiometabolic chaos (5,6).

Controversies persist: does hyperandrogenism drive visceral fat accumulation, or does IR ignite this vicious cycle? (7,8). Asian-specific data is scant, necessitating precise quantification of fat deposition to unravel PCOS pathogenesis. This study dissects body composition in Indian women, probing fat distribution's nexus with metabolic and hormonal havoc. By illuminating adipose anomalies, we aim to forge fat-focused clinical strategies, mitigating IR and mastering metabolic mileau in PCOS.

MATERIALS AND METHODS

This cross-sectional study included 133 women with PCOS and 140 age-matched healthy controls (18–45 years) recruited from the Department of Endocrinology at a tertiary care centre in Southern India (2021–2023).PCOS was diagnosed using revised Rotterdam criteria.

Measurements

Anthropometry: Weight, height, waist/hip circumference, and blood pressure were recorded. BMI, WHR, and body fat percentage (BFP) were calculated.Body composition: Assessed via bioimpedance (Tanita BC-418) during the follicular phase.Biochemical assays: Fasting glucose, insulin (HOMA-IR, QUICKI), and hormones (LH, FSH, testosterone) were analyzed.

Statistical Analysis

Data normality was tested (Kolmogorov-Smirnov). Group comparisons used t-tests (normal distribution) or Wilcoxon rank-sum tests (non-normal). Subgroup analyses by WHR (<0.85 vs. ≥0.85) and BFP (<35% vs. ≥35%) were performed. Linear regression explored body composition's metabolic/hormonal impact (p<0.05 significant). Statistical analysis was performed using SPSS Version 29.

RESULTS

The study compared 133 women with polycystic ovary syndrome (PCOS) and 140 healthy controls across various anthropometric, metabolic, and hormonal parameters. Both groups had similar mean age (28.5 ± 4.3 vs. 29.1 ± 5.0 years, p=0.18) and height (160.2 ± 5.1 vs. 161.0 ± 5.4 cm, p=0.22), the PCOS cohort exhibited significantly elevated adiposity markers. Women with PCOS had significantly higher weight (69.23 ± 12.12 kg vs. 62.8 ± 11.28 kg, P< 0.001), body mass index (BMI) (28.13 ± 4.17 kg/m² vs. 23.62 ± 3.68 kg/m², P< 0.001), and waist circumference (88.13 ± 11.6 cm vs. 81.23 ± 11.6 cm, P < 0.001) compared to controls. Similarly, hip circumference (104.12 ± 7.33 cm vs. 97.84 ± 7.51 cm, P < 0.001) and waist-to-hip ratio (0.83 ± 0.06 vs. 0.71 ± 0.06, P < 0.001) were elevated in the PCOS group. Body composition analysis revealed significantly greater total body fat mass (28.44 ± 11.14 kg vs. 22.33 ± 9.23 kg, P < 0.001), body fat percentage (37.56 ± 8.56% vs. 32.8 ± 7.91%, P < 0.001), and trunk fat mass (16.31 ± 5.39 kg vs. 12.83 ± 4.91 kg, P < 0.001) in PCOS patients.

Metabolic Parameters : Fasting plasma glucose levels did not differ significantly between groups (P = 0.522). However, fasting insulin (13.98 µU/mL vs. 8.9 µU/mL, P < 0.001), HOMA-IR (3.30 vs. 2.06, P < 0.001), and HOMA-B (174 vs. 107, P < 0.001) were significantly higher in the PCOS group, indicating greater insulin resistance. The QUICKI index, reflecting insulin sensitivity, was also lower in PCOS patients (0.320 vs. 0.35, P < 0.001).

Hormonal Profile :Women with PCOS exhibited significantly higher luteinizing hormone (LH) levels (7.0 mIU/mL vs. 4.2 mIU/mL, P < 0.001) and an elevated LH/FSH ratio (1.12 vs. 0.72, P < 0.001). Total testosterone (1.02 nmol/L vs. 0.69 nmol/L, P < 0.001) and androstenedione (11.6 nmol/L vs. 7.1 nmol/L, P < 0.001) were markedly higher in PCOS patients, confirming hyperandrogenism. Conversely, prolactin levels were lower in the PCOS group (11.0 ng/mL vs. 12.7 ng/mL, P = 0.018), while FSH and estrogen levels did not differ significantly (P > 0.05).

Women with PCOS demonstrated significant differences in body composition, insulin resistance, and androgen levels compared to healthy controls. These findings underscore the metabolic and hormonal dysregulation characteristic of PCOS, highlighting the need for targeted interventions addressing obesity, insulin resistance, and hyperandrogenism in this population.

Comparison of Body Composition in PCOS vs. Controls After Waist-to-Hip Ratio Stratification

The study stratified participants into central obesity (WHR ≥ 0.85) and non-central obesity (WHR < 0.85) groups to assess differences in body composition between PCOS patients and controls.

Central Obesity Group (WHR ≥ 0.85) : No significant differences were observed in BMI (30.0 ± 4.5 vs. 29.1 ± 3.1, P = 0.175), waist circumference (97.0 ± 11.0 cm vs. 94.2 ± 6.6 cm, P = 0.071), hip circumference (106.0 ± 9.1 cm vs. 104.8 ± 6.3 cm, P = 0.379), or fat distribution measures between PCOS and control groups.

The waist-to-hip ratio (WHR) was slightly but significantly higher in PCOS women (0.91 ± 0.05 vs. 0.90 ± 0.04, P = 0.041), suggesting more pronounced central fat accumulation. Total body fat mass, body fat percentage, trunk fat mass, and trunk fat percentage did not differ significantly (P > 0.05).

Non-Central Obesity Group (WHR < 0.85): PCOS patients had significantly higher BMI (25.5 ± 4.8 vs. 22.9 ± 4.0, P < 0.001), waist circumference (81.0 ± 8.5 cm vs. 75.5 ± 9.3 cm, P < 0.001), and hip circumference (100.5 ± 8.8 cm vs. 96.5 ± 8.3 cm, P = 0.003) compared to controls. Fat distribution was markedly different, with PCOS women exhibiting greater total body fat mass (23.5 ± 9.3 kg vs. 18.6 ± 7.6 kg, P < 0.001), body fat percentage (35.0 ± 7.5% vs. 30.4 ± 7.0%, P < 0.001), and trunk fat mass (12.5 ± 5.4 kg vs. 9.6 ± 4.7 kg, P < 0.001).

The trunk-to-extremities fat ratio was also higher in PCOS (1.09 ± 0.17 vs. 1.00 ± 0.21, P = 0.002), indicating a tendency toward central fat deposition even in the absence of overt central obesity. In women with central obesity (WHR ≥ 0.85) PCOS and controls had similar body composition, except for a marginally higher WHR in PCOS. In the non-central obesity group (WHR < 0.85), PCOS patients exhibited significantly higher adiposity, particularly in trunk fat, suggesting that PCOS is associated with altered fat distribution even in leaner individuals.

These findings highlight that PCOS influences body composition beyond just central obesity, reinforcing the need for early metabolic risk assessment in all PCOS patients, regardless of WHR classification.

DISCUSSION

This comprehensive study provides critical insights into the intricate relationship between body fat distribution, metabolic dysfunction, and hormonal imbalances in women with polycystic ovary syndrome (PCOS). Our findings reveal significant differences in adiposity patterns between PCOS patients and healthy controls, with profound implications for understanding the pathophysiology and clinical management of this complex endocrine disorder.

Body Composition Abnormalities in PCOS

Our study demonstrates that women with PCOS exhibit distinct body fat distribution patterns regardless of their BMI status. While the higher prevalence of central obesity (53.6% vs 31.7%) in PCOS patients aligns with existing literature (9), our study makes the novel observation that even PCOS women with normal WHR values (<0.85) show significantly greater total body fat (34.9% vs 30.3%) and trunk fat accumulation (35.0% vs 29.3%) compared to controls. This finding challenges the conventional reliance on WHR as the sole indicator of metabolic risk in PCOS and suggests the presence of intrinsic abnormalities in fat partitioning that extend beyond simple anthropometric measures.(10)

The body fat percentage analysis further reinforces this concept, revealing that 70.5% of PCOS patients met criteria for obesity (BFP ≥35%) compared to only 50.4% of controls. Importantly, this adiposity difference persisted across all subgroups, including those with normal BMI, indicating that traditional weight classification systems may fail to capture the true metabolic burden in many PCOS patients(11). These observations support the growing recognition of PCOS as a condition characterized by dysfunctional adipose tissue biology, with potential implications for both metabolic and reproductive dysfunction.

Metabolic Dysregulation Across PCOS Phenotypes : Our results demonstrate consistent metabolic impairment across all PCOS subgroups, with 58.4% of patients meeting criteria for insulin resistance compared to 32.4% of controls. The finding that HOMA-IR values remained elevated in PCOS patients regardless of WHR or BFP status (2.49 vs 1.44 in non-central obese subgroups; 4.08 vs 2.90 in high BFP subgroups) provides compelling evidence that insulin resistance represents a fundamental feature of PCOS pathophysiology rather than simply a consequence of obesity(12).

The regression analysis offers particularly valuable insights, showing that each 1% increase in BFP had a 52% greater impact on HOMA-IR in PCOS patients (β=0.178) compared to controls (β=0.117). This differential susceptibility to adiposity-induced metabolic dysfunction suggests the presence of intrinsic defects in insulin signaling pathways in PCOS that are exacerbated by, but not solely dependent on, excess body fat. Figure 1,(13)

Hormonal Profile and Adiposity Interactions: The endocrine evaluation revealed characteristic PCOS features including elevated LH levels (7.03 vs 4.23 mIU/mL), increased LH/FSH ratios (1.12 vs 0.72), and higher androgen concentrations (testosterone 1.02 vs 0.69 nmol/L). Notably, these hormonal disturbances remained significant across all adiposity subgroups, confirming that neuroendocrine dysregulation represents a core component of PCOS pathophysiology independent of body composition.(14)

However, the observed inverse relationship between BFP and both LH (β=-0.135) and androstenedione (β=-0.151) levels in PCOS patients introduces an important nuance to our understanding of obesity's role in PCOS manifestations. This finding aligns with previous reports of attenuated LH secretion in obese PCOS women and suggests that excess adiposity may partially mitigate gonadotropin abnormalities through mechanisms potentially involving insulin-mediated suppression of GnRH pulsatility or enhanced steroid negative feedback.(15)

CONCLUSION

This study underscores that PCOS is associated with altered body composition, insulin resistance, and hormonal dysregulation, independent of traditional obesity markers. Even non-obese PCOS women exhibit increased adiposity and metabolic dysfunction, reinforcing the need for universal metabolic screening in all PCOS patients.

Table 1. Baseline Characteristics:

Characteristic	PCOS	Control	P value
	(n=133)	(n=140)	
	(Mean ± SD)	(Mean ± SD)	
Age, y	27.32 ± 3.80	28.42 ± 6.33	0.987
Height, m	1.57 ± 0.06	1.59 ± 0.03	0.336
Weight, kg	69.23 ± 12.12	62.8 ± 11.28	<0.001
Body mass index, kg/m2	28.13 ± 4.17	23.62 ± 3.68	<0.001
Waist circumference, cm	88.13 ± 11.6	81.23 ± 11.6	<0.001
Hip circumference, cm	104.12 ± 7.33	97.84 ± 7.51	<0.001
Waist-to-hip ratio	0.83 ± 0.06	0.71 ± 0.06	<0.001
Total body fat mass, kg	28.44 ± 11.14	22.33 ± 9.23	<0.001
Body fat percentage, %	37.56 ± 8.56	32.8 ± 7.91	<0.001
Trunk fat mass, kg	16.31 ± 5.39	12.83 ± 4.91	<0.001
Trunk fat percentage, %	39.32 ± 9.33	33.39 ± 9.87	<0.001
Fasting plasma glucose, (mg/dl)	95.5 (84.7; 104.5)	93.7 (88.3; 100.9)	0.522
Fasting insulin, (μU/ml)	13.98 (9.8;22.09)	8.9 (5.1;14.32)	<0.001
HOMA-IR	3.30 (2.05; 5.70)	2.06 (1.11;3.56)	<0.001
HOMA-B	174 (118.35;257.92)	107.33 (72.83;157.8)	<0.001
QUICKI	0.321(0.31;0.33)	0.35 (0.31;0.38)	<0.001
Prolactin (ng/mL)	11.0 (8.6; 14.7)	12.7 (9.8; 17.6)	0.018
FSH (mIU/mL)	5.8 (4.8; 7.0)	6.1 (5.1; 7.1)	0.299
LH (mIU/mL)	7.0 (3.7; 9.5)	4.2 (3.2; 5.2)	<0.001
LH/FSH Ratio	1.12 (0.67; 1.75)	0.72 (0.47; 0.88)	<0.001
Estrogen (pmol/L)	180 (138; 226)	169 (143;204)	0.168
Total Testosterone (nmol/L)	1.02 (0.69; 1.49)	0.69 (0.69; 0.81)	<0.001
Androstenedione (nmol/L)	11.6 (7.3; 16.2)	7.1 (5.4; 9.6)	<0.001

Data expressed as mean ± SD,median (IQR)
HOMA-IR: homeostasis model assessment of insulin resistance;
HOMA-B: homeostasis model assessment of b-cell function;
QUICKI: quantitative insulin sensitivity check index.

Table 2 Comparison of Body Composition Indices

Body composition indicators	Central obesity			Non-central obesity		
	PCOS (n=73)	Control (n=46)	P value	PCOS (n=60)	Control (n=94)	P value
Body mass index, kg/m²	30.0 ± 4.5	29.1 ± 3.1	0.175	25.5 ± 4.8	22.9 ± 4.0	<0.001
Waist circumference,cm	97.0 ± 11.0	94.2 ± 6.6	0.071	81.0 ± 8.5	75.5 ± 9.3	<0.001
Hip circumference,cm	106.0 ± 9.1	104.8 ± 6.3	0.379	100.5 ± 8.8	96.5 ± 8.3	0.003
Waist-to-hip ratio	0.91 ± 0.05	0.90 ± 0.04	0.041	0.80 ± 0.03	0.78 ± 0.04	<0.001
Total body fat mass, kg	33.0 ± 10.8	30.6 ± 6.8	0.139	23.5 ± 9.3	18.6 ± 7.6	<0.001
Body fat percentage, %	41.6 ± 6.2	40.7 ± 4.2	0.355	35.0 ± 7.5	30.4 ± 7.0	<0.001
Trunk fat mass, kg	18.1 ± 6.2	16.9 ± 3.9	0.183	12.5 ± 5.4	9.6 ± 4.7	<0.001
Trunk fat percentage, %	43.1 ± 7.6	42.2 ± 5.1	0.405	35.0 ± 9.3	29.4 ± 8.9	<0.001
Trunk-to-extremities fat ratio	1.21 ± 0.12	1.24 ± 0.12	0.251	1.09 ± 0.17	1.00 ± 0.21	0.002

[a]The distribution was expressed as mean ± standard deviation, and a bilateral t-test was applied. P-values with statistical differences (P < 0.05) have been bolded.

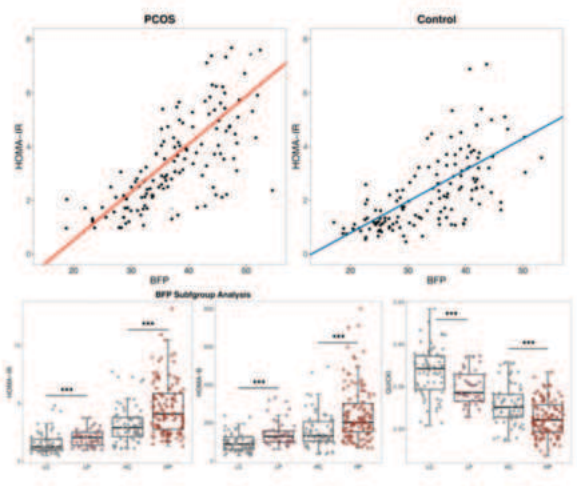

Figure 1: Correlation Indices and BFP Subgroup Analysis.

BFP: Body fat percentage, HP, High body fat percentage PCOS group; HC, High body fat percentage control group; LP, Low body fat percentage PCOS group; LC, Low body fat percentage control group

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