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Body Fat and Hormonal Imbalances in Polycystic Ovary Syndrome: A Comparative Analysis of Treatment Outcomes

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ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) is a common endocrine disorder linked to obesity, insulin resistance, and reproductive dysfunction. Despite various treatments, hormonal imbalances persist, complicating management.

Objective: This study investigates the anthropometric and hormonal profiles of PCOS patients, focusing on LH, FSH, and body composition in treated and untreated groups, compared to healthy controls.

Methods: A total of 30 PCOS patients (15 treated, 15 untreated) and 20 healthy controls were assessed for BMI, body fat percentage, and serum LH/FSH levels using ELISA. Statistical analysis included t-tests and correlation analyses.

Results: PCOS patients had significantly higher LH and lower FSH levels than controls ($p<0.05$), with no difference between treated and untreated groups. Body fat was elevated in both PCOS groups but did not correlate with hormonal changes. BMI correlated with body fat in controls ($r=0.6$, $p<0.01$), but not in PCOS patients.

Conclusion: Hormonal imbalances in PCOS persist despite treatment. Body fat may trigger, but not regulate, PCOS. Personalized therapies targeting deeper mechanisms are needed.

Keywords: *PCOS; BMI; LH/FSH ratio; body fat; hormonal imbalance; personalized therapy.*

1. INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common endocrine disorder affecting 5%–10% of women of reproductive age worldwide, and it is strongly associated with obesity and metabolic dysfunctions. Obesity, particularly central adiposity, exacerbates the clinical and metabolic features of PCOS, making it a crucial factor in the severity of the syndrome (1,2). The relationship between obesity and PCOS is bidirectional, where obesity can worsen hyperandrogenism, insulin resistance, and ovulatory dysfunction, while PCOS may predispose individuals to greater fat accumulation and difficulty in weight management (3) Body mass index (BMI) and basal metabolic rate (BMR) are key parameters in assessing and managing obesity in PCOS, as both influence the metabolic profile and overall health outcomes of patients.

Women with PCOS are more likely to have elevated BMI, particularly abdominal obesity, which is a significant contributor to insulin resistance. Increased fat mass, especially in the visceral region, leads to an overproduction of adipokines, pro-inflammatory cytokines, and free fatty acids, all of which impair insulin signalling and glucose metabolism (4) This exacerbates insulin resistance, a hallmark of PCOS, further promoting hyperandrogenism and menstrual irregularities. Furthermore, obesity in women with PCOS is often associated with a decreased basal metabolic rate (BMR), which impedes weight loss efforts and contributes to a cycle of worsening metabolic and reproductive health (5).

BMI serves as a critical marker in clinical settings for assessing the extent of obesity in PCOS patients. Studies have shown that higher BMI is linked to more severe insulin resistance and a higher likelihood of metabolic syndrome in these individuals, increasing their risk of developing type 2 diabetes and cardiovascular diseases (6). Importantly, even modest reductions in BMI through lifestyle interventions such as diet and exercise have been shown to improve insulin sensitivity, ovulatory function, and reproductive outcomes in women with PCOS (7). Therefore, BMI is not only a diagnostic tool but also a target for intervention in the management of PCOS (5)

Basal metabolic rate (BMR), which accounts for the energy expenditure at rest, is often reduced in individuals with PCOS, especially those with obesity. A lower BMR may contribute to the difficulty in achieving weight loss, as women with PCOS require a greater caloric deficit to achieve the same weight loss as non-PCOS individuals (8) This reduced BMR may result from alterations in muscle mass, mitochondrial function, and metabolic efficiency in individuals with PCOS. Therapeutic approaches aimed at increasing BMR through resistance training, aerobic exercise, and dietary modifications have been shown to be beneficial in improving weight management and metabolic health in PCOS patients (9)

The aim of this study was to evaluate anthropometric, clinical, and hormonal imbalance (LH and FSH levels, LH:FSH ratio) in PCOS subgroups and healthy controls, focusing on oxidative stress and hormonal imbalance. The objectives included assessing body composition, body fat percentage, and the prevalence of hypertension or hypotension among untreated and treated PCOS females.

2. METHODOLOGY

This study was approved by Midnapore City College, Midnapore, West Bengal. A brief explanation of the study and informed written consent were obtained from all participants. The study involved two groups of female students aged 18-25 diagnosed with PCOS. The first group comprised 22 students previously diagnosed with PCOS who had not received treatment, while the second group included 22 students undergoing treatment for PCOS. Additionally, 28 age-matched healthy females were enrolled as healthy controls (HCs). Participants were randomly selected, and a survey form (either in hard copy or digital format) was provided to ensure appropriate subject selection under the study criteria.

The diagnosis of PCOS was based on the Rotterdam ESHRE/ASRM-sponsored PCOS consensus criteria (2003), requiring patients to meet at least two of the following: a) anovulation or oligomenorrhea, b) clinical or biochemical evidence of hyperandrogenism, and c) the presence of polycystic ovaries on ultrasound. Participants with hematological or other malignancies, as well as those with a history of smoking or alcohol use, were excluded. Additionally, individuals with associated disorders such as Cushing's disease, hypothyroidism, hyperprolactinemia, adrenal hyperplasia, or ovarian tumours were not included in the study. Patients lacking medical documentation to confirm PCOS diagnosis were also excluded.

2.1 Measurement of Anthropometric Parameters

The study data were measured using an Omron HBF 375 Karada Scan Body Composition Monitor–Body Fat analyzer to analyse body composition (10) .

Height- Height was measured by using the anthropometric rod, in centimetres. Weight- Weight is measured by using a weight machine, in kg one of the most important measurements in nutritional assessment is body weight. BMI- BMI was calculated from height from height and weight, using the following equation. (11).

$$BMI = \frac{\text{Weight(kg)}}{\text{Hight(m)2}}$$

BSA- BSA is calculated from height and weight using the following equation.

$$\text{BSA} = \sqrt{\frac{[Height(cm) \times Weight(kg)]}{3600}}.$$

Waist hip ratio- This ratio was determined by using measuring tape to determine the circumference of hips at the widest part of the buttocks. Waist circumference was measured midway between the lowest rib margin and the iliac crest. The ratio is calculated by dividing the waist measurement by the hip measurement (11)

$$\text{Waist Hip Ratio} = \frac{Waist\ Circumference}{Hip\ Circumference}.$$

Total body fat – Total body fat is calculated from waist, hip, and neck circumference. Equation of total body fat (12).

Body fat percentage = $163.205\times log10(waist + hip - neck) - 97.684 \times \log10$ (height)-78.387. Basal metabolic rate: BMR was measured using the method of Johnstone et al., 2005. (13).

2.2 Blood pressure measurement-

Blood pressure was measured by auscultatory method using a stethoscope and sphygmomanometer. (14)

2.3 Sample collection:

A total of 5 mL of venous blood samples were obtained through plastic injectors from the antecubital vein by a specialized lab technician. Within 30 minutes after being drawn, the blood samples were centrifuged at 3,000 rpm for 10 minutes. The supernatant serum samples were placed in plastic tubes. All the analysis should be done immediately just after the collection of samples.

2.4 Determination of Luteinizing Hormone (LH) and Follicle Stimulating Hormone (FSH) by Sandwich ELISA

Serum LH and FSH levels were assessed by ELISA using Human FSH ELISA Kit -LS- F20633 reagents and Human LH ELISA Kit -LS-F27048 respectively. The test was done according to the manufacturers' kit protocol.

2.5 Statistical analysis

All statistical analyses were performed using GraphPad Prism v 5.0, (GraphPad Software INC, CA, USA) and MedcalC v 11.6, (Belgium). All normally distributed data were tested by applying the Shapiro-Wilk test. Normally distributed variables were presented as mean values with standard deviation (SD). The difference between groups was estimated by the independent *t*test and Man-Whitney *U*test for normally distributed and skewed data, respectively. Spearman (r) value was used for the correlation coefficient. All *p*values <0.05 were considered statistically significant.

3. RESULT

3.1 Assessment of serum concentration of LH and FSH

Data from the serum concentration of LH and FSH and LH/FSH ratio showed significant alteration. The serum concentration of LH was significantly upregulated in the patient's group irrespective of the untreated or treated group, compared to the healthy control (*p=0.005, and p=0.02* respectively). No significant alteration was observed in the concentration of LH between the two patient groups (*p=0.4*) **(Fig: 1A).**

The serum concentration of FSH is significantly lower in the patient group than in the HC group (*p=0.004, and p=0.01* untreated and treated group respectively). The patient groups didn't show any significant differences between them (*p=0.2*) **(Fig: 1B).**

Compared to the untreated group the ratio between the serum concentration of LH/FSH also showed a significant higher concentration irrespective of the patient group (*p=0.0008, and p=0.001* respectively). Alteration in the serum concentration between the patient groups was not observed (*p=0.8*) **(Fig: 1C).**

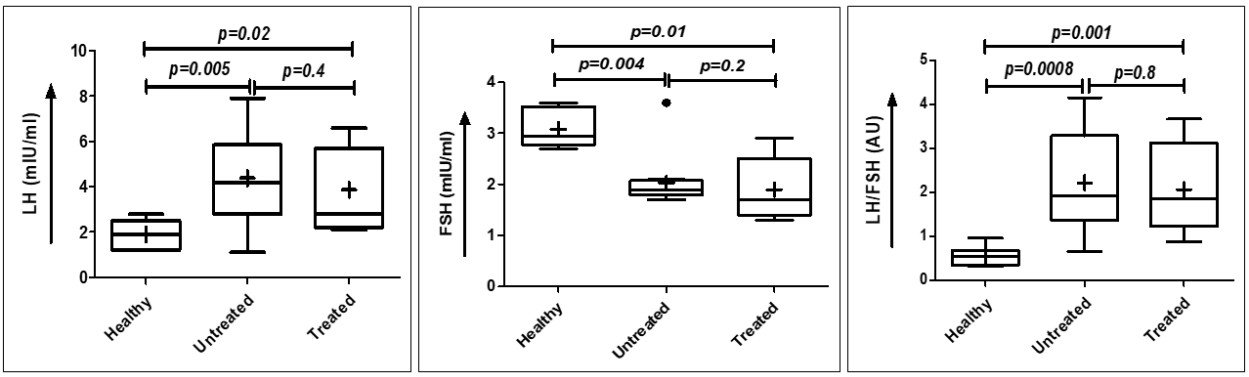

Fig. 1: Comparison of serum LH, FSH, and LH/FSH ratio across groups.

(A) Graph illustrating serum LH levels for control, treated, and untreated patient groups. **(B)** Graph showing serum FSH levels across control and patient groups. **(C)** Representation of the LH/FSH ratio between control, treated, and untreated groups.

3.2 Measurement of the anthropometric and physiological parameters

We also assessed the anthropometric and physiological parameters. Pulse pressure was significantly upregulated irrespective

of the patient groups (*p=0.002 and p=0.01* untreated and treated groups respectively). Untreated and treated patient groups didn't show a significant difference (*p=0.8*) **(Fig: 2A).**

Compared to the HC, total body fat percentage (%) was significantly higher in both treated and untreated groups (*p=0.002, and p=0.006* respectively). A similar percentage of body fat was observed within the patient groups (*p=0.9*) **(Fig: 2B).** Pulse rate was found to be elevated in patient participants irrespective of the treatment (*p=0.7*). Both of the patient groups showed elevation in the pulse rate compared to the untreated participants (*p=0.005 and p=0.02* untreated and treated groups respectively) **(Fig: 2C).**

Similar trends were followed by BMR. HCs showed lower BMR compared to both of the patient groups (*p=0.0006 and p=0.0001* untreated and treated groups respectively). No statistical significance was observed regarding the BMR, in-between the patient groups (*p=0.5*) **(Fig: 2D).** No significant statistical differences were observed irrespective of all groups **(Fig: 2E).**

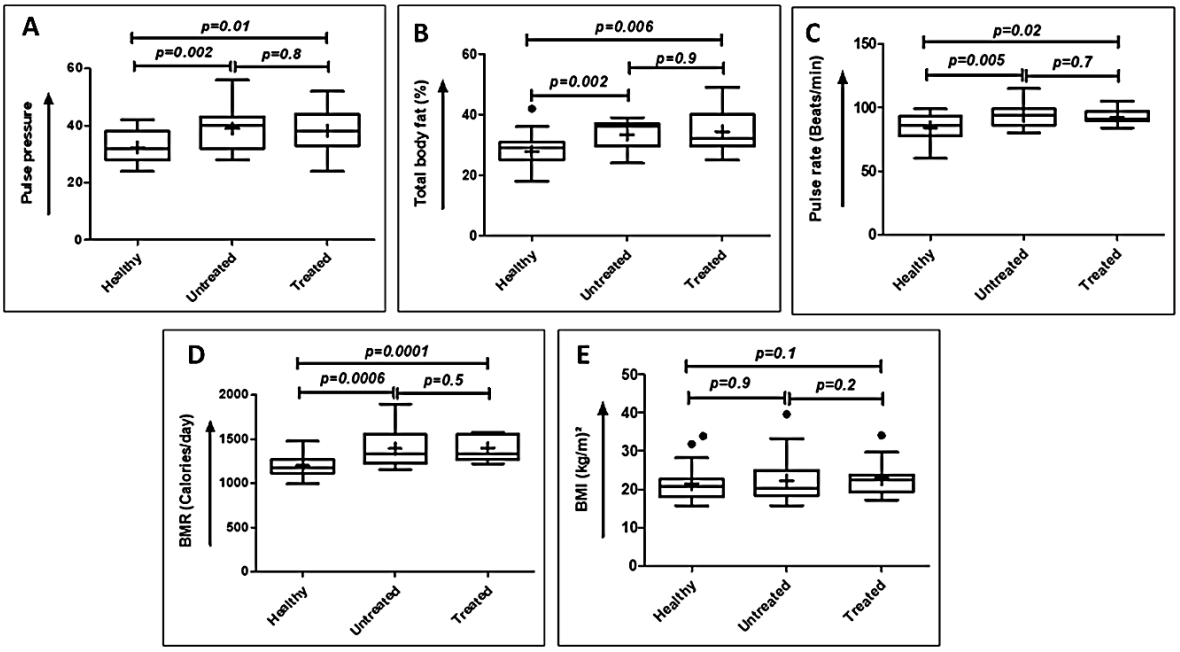

Fig. 2: Analysis of key anthropometric and physiological parameters

(A) Graph displaying pulse pressure across control, treated, and untreated groups. **(B)** Bar chart comparing total body fat percentage among all groups. **(C)** Graph showing pulse rate across the control, treated, and untreated groups. **(D)** Comparison of basal metabolic rate (BMR) between control and patient groups. **(E)** Graph showing body mass index (BMI) across all groups.

3.3 Correlation analysis

The correlation analysis was done between the BMI, and BMR with the total body fat percentage (%). BMI showed a significant positive correlation with the total body fat percentage in the case of HCs (*r= 0.6 p=0.0003*) **(Fig 3A).** No such significant association was observed for the same in the case of patient participants irrespective of the group based on treatment (*r= 0.3 p=0.1* irrespectively) **(Fig 3B and 3C).**

A similar association was observed for BMR. HCs showed a positive significant association between BMR and total body fat percentage (*r= 0.5 p=0.01*) **(Fig 3D).** No significant association was observed in the untreated and treated group for the same. (*r= 0.07 p=0.7 and r= -0.3 p=0.4 irrespectively*) **(Fig 3E and 3F).**

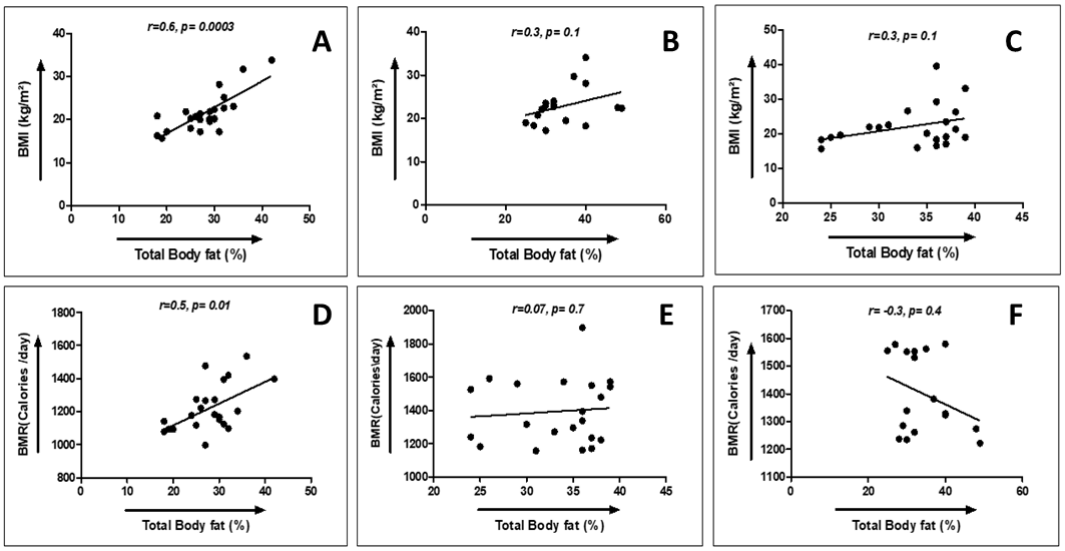

Fig. 3: Correlation analysis between BMI, BMR, and total body fat percentage

(A) Scatter plot illustrating the relationship between BMI and total body fat percentage in healthy controls. **(B-C)** Scatter plots showing the correlation between BMI and total body fat percentage in treated and untreated patient groups. **(D)** Scatter plot showing the relationship between BMR and total body fat percentage in healthy controls. **(E-F)** Scatter plots illustrating the correlation between BMR and total body fat percentage in treated and untreated patient groups.

4. DISCUSSION

Polycystic Ovarian Syndrome (PCOS), one of the most common endocrine disorders affecting women, has widespread implications on fertility, metabolism, and overall health (15,16) The disorder is characterized by a combination of hyperandrogenism, ovulatory dysfunction, and polycystic ovaries, and is strongly associated with metabolic disturbances such as insulin resistance and obesity (17) Despite advances in understanding its pathophysiology, the effectiveness of current treatment regimens remains controversial, especially concerning hormonal modulation (5,6). Our study brings into focus critical questions about the efficacy of current therapeutic strategies for PCOS. We found significant upregulation of luteinizing hormone (LH) and downregulation of follicle-stimulating hormone (FSH) in both treated and untreated patient groups, with no significant inter-group differences. The elevated LH/FSH ratio, a well-established diagnostic marker for PCOS, similarly showed no significant variation between treated and untreated groups. This lack of hormonal modulation in response to treatment challenges the effectiveness of current therapeutic approaches in addressing the core endocrine abnormalities in PCOS, a finding consistent with more recent studies that have reported persistent hormonal imbalances in PCOS patients despite conventional therapies (18,19). The persistent hormonal dysregulation seen in our study aligns with current understanding of PCOS, where elevated LH and reduced FSH contribute to disrupted ovarian function, anovulation, and infertility (17,20). This imbalance is also closely tied to insulin resistance and hyperandrogenism, two key features of PCOS (8,21). However, the continued presence of these abnormalities in both treated and untreated groups suggests that current treatments typically involving oral contraceptives, insulin sensitizers, and anti-androgenic agents are not adequately addressing the underlying pathophysiological mechanisms. Previous research has also indicated that these treatments mainly provide symptomatic relief rather than targeting the endocrine disturbances at the root of PCOS (6,22), which is corroborated by our findings. A particular concern is the lack of significant differences in the LH/FSH ratio between treated and untreated groups, raising doubts about the effectiveness of treatment regimens in modulating these hormones. A study by Qiao & Feng (2020) also reported no significant impact of treatment on the LH/FSH ratio, suggesting that conventional therapies may need to be reevaluated (18). This further reinforces the need for individualized treatment strategies that account for the specific hormonal and metabolic profiles of patients, which may offer better outcomes (6,23). In exploring body fat as a potential contributing factor to PCOS, our study found a significant positive correlation between total body fat percentage, BMI, and basal metabolic rate (BMR) in healthy control groups, consistent with recent findings (24–26). However, this correlation was absent in both treated and untreated PCOS patient groups, suggesting that while body fat is a triggering factor for PCOS development, it may not serve as a primary regulator once the disorder is established. This contradicts earlier studies that emphasized the role of adiposity in driving PCOS progression and response to treatment (Lim et al., 2019). Our data implies that while reducing body fat can alleviate some PCOS symptoms, it may not directly correct the hormonal imbalances central to the disorder (8). These findings open new avenues for further research. Future investigations should focus on alternative pathways, such as the roles of chronic inflammation, oxidative stress, and epigenetic modifications in PCOS pathogenesis. Emerging evidence suggests that these systemic factors may be more critical than previously thought

in maintaining hormonal dysregulation and contributing to the persistence of symptoms, even in patients receiving treatment (8,17) In light of this, clinical management of PCOS should shift towards personalized medicine, focusing on tailoring treatments to individual hormonal and metabolic profiles. Recent clinical guidelines advocate for such customized therapeutic interventions in managing PCOS phenotypes (6,26).

Table 1: Brief demography of the study participants.

Variables	Healthy Control (n= 28) (Mean ±SD)	Untreated PCOS (n= 22) (Mean ±SD)	Treated PCOS (n= 22) (Mean ±SD)	ANOVA P -Value	Control vs. Untreated P -Value	Control vs. Treated P-Value	Untreated vs. Treated P -Value
Height(cm)	154.32±6.9	155±6.8	153.9±5.4	NS	NS	NS	NS
Age (Years)	22.6± 2.8	21.8± 2.8	22.9± 2.6	NS	NS	NS	NS
Weight(kg)	51.4±11.7	52.7±12.5	53.6±8.5	NS	NS	NS	NS
BSA (m^2)	1.4±0.1	1.4±0.2	1.5±0.3	NS	NS	NS	NS
Waist-hip ratio	0.8±0.05	0.8±0.04	0.8±0.03	NS	NS	NS	NS

5. CONCLUSION

In conclusion, while body fat appears to trigger the onset of PCOS, it does not seem to regulate the disorder once it is established. Moreover, current treatments do not sufficiently modify the underlying hormonal dysregulation, raising questions about their long-term efficacy. Our study emphasizes the need for more targeted interventions and a move towards personalized treatment strategies that address the complex endocrine and metabolic dimensions of PCOS.

Statement and declaration:

- Conflict of interest: Nothing to declare.
- **Author Contributions:** All authors contributed to the study conception and design and helped in the perusal of the study. The first draft of the manuscript was written by, Dr. Dipanjan Bhattacharjee, and all authors commented on the subsequent versions of the manuscript. All authors read and approved the final manuscript.
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- **Ethical approval:** This study was approved by the Institutional Ethical Committee.
- **Consent to participate:** Written, informed consent was taken from each participant. This study was done following the Declaration of Helsinki, 1964, and its later amendments.
- **Consent to publish:** No individual patient information has been disclosed in the manuscript.

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