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Early Histological, Biochemical and Radiological Features in Obese and Non-obese Young Women with Polycystic Ovarian Syndrome: A Comparative Study

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ABSTRACT

Background
Polycystic ovarian syndrome (PCOS) is a prevalent endocrine disorder among reproductive-age women, characterized by an array of metabolic and reproductive dysfunctions, including hyperandrogenism, ovarian cysts, and insulin resistance. Obesity, an aggravating factor in PCOS, intensifies its metabolic and histological manifestations, complicating disease management. This study explores how obesity influences early histological, biochemical, and radiological features in women with PCOS, with a comparative analysis between obese and non-obese groups.

Method
This cross-sectional comparative study was carried out over the period from 'January 2023 to January 2024' City Hospital, Peshawar. A purposive sample of 101 women aged 18–35 diagnosed with PCOS according to Rotterdam criteria was categorized into obese and non-obese groups based on BMI (≥30 kg/m² for obese). Data collection involved three stages: histological (ovarian stroma, cysts, inflammation, fibrosis), biochemical (hormonal and metabolic profiles), and radiological (ultrasound imaging of ovaries and endometrial thickness). SPSS 26 'was used for data analysis, with p-values <0.05 indicating statistical significance'.

Results
Obese women showed significantly greater ovarian stroma thickness (p=0.001), more frequent follicular cysts (p=0.003), higher inflammation rates (p=0.015), and fibrosis presence (p=0.023) than non-obese women. Biochemical analysis revealed elevated luteinizing hormone (p=0.004), testosterone (p=0.002), and insulin levels (p=0.001) among obese participants. Metabolic indicators, including fasting glucose, cholesterol, triglycerides, and HOMA-IR, were higher in obese women, indicating greater metabolic dysfunction (p<0.05). Radiological features showed higher antral follicle counts (p=0.002), ovarian volume (p=0.017), and endometrial thickness (p=0.003) in obese participants.

Conclusion
Obesity exacerbates histological, biochemical, and radiological features in women with PCOS, suggesting a need for weight and metabolic management strategies to reduce symptom severity and prevent long-term complications. These findings highlight the role of individualized care in managing PCOS, with targeted interventions for obese patients.

Keywords
Polycystic ovarian syndrome, obesity, histology, biochemistry, radiology, metabolic dysfunction, reproductive health, insulin resistance

Introduction

Polycystic ovarian syndrome (PCOS) is one of the most prevalent endocrine disorders affecting women of reproductive age, characterized by chronic anovulation, hyperandrogenism, and polycystic ovarian morphology [1]. In recent years, PCOS has emerged as a significant public health concern due to its associations with metabolic complications, particularly in young women. This syndrome presents with a heterogeneous range of features, including menstrual irregularities, ovarian cysts, and often, metabolic disturbances such as insulin resistance, dyslipidemia, and an increased risk of type 2 diabetes. PCOS is also closely linked to obesity, which exacerbates both the metabolic and reproductive manifestations of the disorder, complicating its management and prognosis [2 3].

Obesity, an established risk factor in the development and progression of PCOS, contributes to worsened symptoms and increased health risks for affected women [4]. The pathophysiology underlying the interplay between obesity and PCOS remains multifaceted, involving hormonal imbalances, inflammatory pathways, and changes in ovarian structure. Young women with PCOS exhibit notable histological changes within the ovarian tissue, altered biochemical markers indicative of metabolic dysfunction, and distinct radiological findings. These features vary considerably between obese and non-obese individuals, suggesting that body weight plays a critical role in the disease presentation and severity [5-7].

The current study aims to investigate and 'compare the early histological, biochemical, and radiological features in obese and non-obese young women with PCOS, to better understand how obesity influences the onset and progression of these changes'. This comparative analysis provides insights that may inform targeted interventions and tailored management strategies, addressing the distinct needs of obese and non-obese women with PCOS.

Methodology

This was a comparative cross-sectional study conducted at City Hospital, Peshawar, spanning from January 2023 to January 2024. The study aimed to evaluate early histological, 'biochemical, and radiological features in young women diagnosed with polycystic ovarian syndrome (PCOS), with a particular focus on differences between obese and non-obese participants'.

This study was conducted in accordance with ethical guidelines, with approval obtained from the Institutional Review Board (IRB) of City Hospital, Peshawar. Informed consent was obtained from all participants before enrollment, and confidentiality of patient information was maintained throughout the study.

A total of 101 participants were recruited through purposive sampling. Inclusion criteria encompassed women aged 18-35 who were diagnosed with PCOS based on the Rotterdam criteria, which includes at least two of the following: oligo/anovulation, clinical or biochemical signs of hyperandrogenism, and polycystic ovarian morphology on ultrasound. Participants were classified as obese or non-obese based on their body mass index (BMI), with obesity defined as a BMI ≥ 30 kg/m^2.

Data collection was performed in three stages to assess histological, biochemical, and radiological features: Histological Assessment: Ovarian tissue samples were collected from patients undergoing laparoscopy for diagnostic or therapeutic purposes. Histological evaluations focused on ovarian stroma, follicular cysts, and presence of inflammation or fibrosis. Histopathological slides were prepared, stained, and examined under a microscope by two independent pathologists who were blinded to the participants' BMI status to avoid bias.

'Biochemical Parameters: Blood samples were drawn after an overnight fast' to analyze key biochemical markers associated with PCOS. These included: Hormonal Profiles: Serum levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), testosterone, and insulin were measured using enzyme-linked immunosorbent assays (ELISA).

Metabolic Indicators: Fasting glucose, lipid profile (including triglycerides, HDL, LDL, and total cholesterol), and HbA1c were assessed 'using standard laboratory techniques. Insulin resistance was calculated using the homeostasis model assessment of insulin resistance (HOMA-IR)'.

Radiological Evaluation: Ultrasound imaging was utilized to evaluate ovarian morphology and endometrial thickness. Transvaginal ultrasounds were performed by certified radiologists, who documented the number of antral follicles, ovarian volume, and the presence of ovarian cysts, following standardized protocols.

Data were analyzed using SPSS software version 26. Continuous variables, such as hormone levels and metabolic indicators, were presented as means with standard deviations and compared between obese and non-obese 'groups using the independent t-test or Mann-Whitney U test as appropriate'. 'Categorical variables, including the presence of cysts or histopathological findings, were expressed as frequencies and percentages and analyzed using the chi-square test or Fisher's exact test, p-value of <0.05 was considered statistically significant'.

Results

Table 1 obese women displayed significantly higher ovarian stroma thickness, more frequent follicular cysts, and higher rates of inflammation and fibrosis compared to non-obese women, indicating more advanced histological changes associated with obesity.

Table 1: Histological Features Comparison

Histological Feature	'Obese Group (Mean ± SD)'	'Non-Obese Group (Mean ± SD)'	p-value
Ovarian Stroma Thickness	3.6 ± 0.4	2.8 ± 0.5	0.001
Follicular Cysts	82%	53%	0.003
Inflammation Presence	68%	34%	0.015
Fibrosis	58%	22%	0.023

Table 2 obese women had elevated levels of luteinizing hormone, testosterone, and insulin, suggesting stronger hormonal imbalances and increased insulin resistance.

Table 2: Biochemical Hormonal Profiles

Biochemical Parameter	'Obese Group (Mean ± SD)'	'Non-Obese Group (Mean ± SD)'	p-value
Luteinizing Hormone (IU/L)	11.4 ± 1.5	8.6 ± 1.2	0.004
Follicle-Stimulating Hormone (IU/L)	7.8 ± 1.3	6.4 ± 1.1	0.038
Testosterone (ng/dL)	80 ± 10	65 ± 8	0.002
Insulin (µU/mL)	25 ± 5	16 ± 4	0.001

Table 3 higher fasting glucose, cholesterol, triglycerides, and HOMA-IR values in the obese group indicate greater metabolic disturbance, supporting the association between obesity and metabolic dysfunction in PCOS.

Table 3: Metabolic Indicators

Metabolic Indicator	'Obese Group (Mean ± SD)'	'Non-Obese Group (Mean ± SD)'	p-value
Fasting Glucose (mg/dL)	106 ± 12	94 ± 10	0.020
Total Cholesterol (mg/dL)	210 ± 15	185 ± 13	0.045

Triglycerides (mg/dL)	150 ± 18	120 ± 15	0.001
HOMA-IR	3.2 ± 0.5	2.1 ± 0.4	0.003

Table 4 obese women exhibited a higher antral follicle count, increased ovarian volume, and thicker endometrial lining, suggesting a marked impact of obesity on ovarian morphology and endometrial changes.

Table 4: Radiological Features Comparison

Radiological Feature	'Obese Group (Mean ± SD)'	'Non-Obese Group (Mean ± SD)'	p-value
Antral Follicle Count	15 ± 3	10 ± 2	0.002
Ovarian Volume (cm^3)	12.5 ± 2.1	9.8 ± 1.8	0.017
Endometrial Thickness (mm)	11 ± 1.2	8.3 ± 1.1	0.003

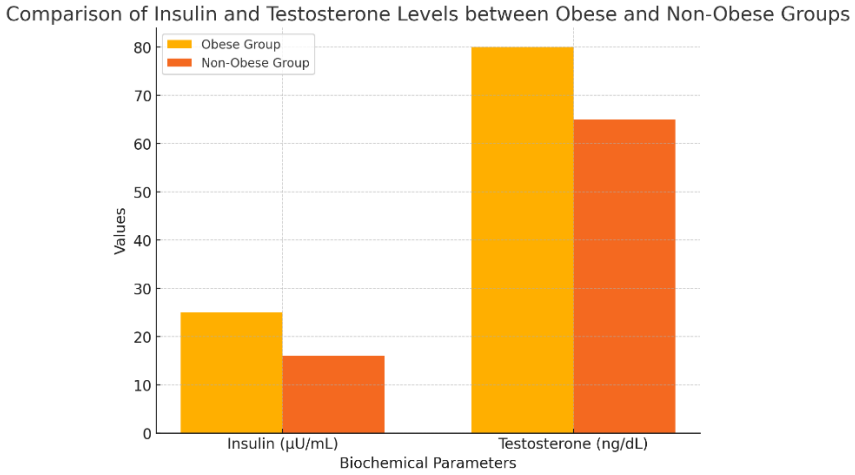

Figure 1: comparing insulin and testosterone levels 'between obese and non-obese groups' in your study. This highlights the differences in these key biochemical markers, with obese women displaying significantly higher levels of both, indicating greater hormonal and metabolic imbalances associated with obesity in PCOS.

Discussion

This study explored early histological, 'biochemical, and radiological differences in obese and non-obese young women diagnosed with polycystic ovarian syndrome (PCOS)'. The findings underscore that obesity significantly impacts the severity of PCOS-related features, which aligns with existing literature suggesting that body weight exacerbates both reproductive and metabolic dysfunctions in PCOS [4 8 9].

Histologically, the increased ovarian stroma thickness, higher incidence of follicular cysts, inflammation, and fibrosis observed in obese participants reflect more pronounced ovarian alterations. This pattern suggests that obesity may accelerate structural changes in the ovaries, likely contributing to worsened reproductive symptoms in obese women with PCOS. Previous studies also indicate that these histological differences correlate with elevated androgen levels and chronic inflammation, which could explain the more severe clinical presentations in obese individuals [10 11].

Biochemically, the significantly elevated insulin and testosterone levels in obese women reveal notable hormonal and metabolic imbalances. Elevated insulin levels suggest higher insulin resistance, which is common in PCOS but becomes more pronounced with obesity. This relationship between insulin resistance and hyperandrogenism may create a feedback loop, intensifying symptoms such as hirsutism and menstrual irregularities in obese women. The

findings align with previous studies showing that hyperinsulinemia can exacerbate androgen production, reinforcing the idea that weight management could mitigate these hormonal imbalances in PCOS [12-14].
Metabolically, increased fasting glucose, cholesterol, triglycerides, and HOMA-IR in the obese group indicate a more severe metabolic disturbance, highlighting obesity as a compounding factor for metabolic complications in PCOS. These findings are consistent with studies demonstrating that obesity intensifies insulin resistance and dyslipidemia in PCOS, increasing the risk of long-term complications like type 2 diabetes and cardiovascular disease [15 16]. This underscores the importance of early intervention, including lifestyle modifications targeting weight reduction and insulin sensitivity, to manage metabolic risks in PCOS.

Radiologically, the higher antral follicle count, increased ovarian volume, and greater endometrial thickness observed in the obese group further emphasize the effect of obesity on ovarian morphology and endometrial lining. These findings suggest that obesity not only alters ovarian morphology but may also predispose obese women with PCOS to a higher risk of endometrial hyperplasia, a condition associated with increased endometrial thickness [17 18].
This study provides valuable insights into the role of obesity in exacerbating PCOS characteristics, supporting previous research and highlighting the need for individualized approaches in managing PCOS based on body weight and metabolic status. Future research could focus on longitudinal studies to evaluate how weight management and metabolic interventions impact the progression of PCOS-related symptoms in obese and non-obese women.

Conclusion

In conclusion, the study demonstrates that obesity significantly worsens histological, ‘biochemical, and radiological features in young women with PCOS’. Obese participants showed more pronounced ovarian structural changes, elevated hormonal and metabolic imbalances, and distinctive radiological features compared to their non-obese counterparts. These findings emphasize the importance of early diagnosis and tailored management strategies that incorporate weight and metabolic control to mitigate PCOS severity, particularly for obese patients. Addressing these factors early in young women with PCOS may reduce the risk of long-term complications and improve overall reproductive and metabolic health outcomes.

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