Original Research

COMPARISON OF CLINICAL, METABOLIC AND ENDOCRINAL PARAMETERS OF OBESE AND LEAN POLYCYSTIC OVARY SYNDROME PATIENTS – CROSS-SECTIONAL ANALYTICAL STUDY

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ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS)-recently recognized by medical organizations as Polyendocrine Metabolic Ovarian Syndrome (PMOS)-is a common endocrine disorder affecting women of reproductive age and is frequently associated with obesity, which may aggravate its clinical, metabolic, and hormonal manifestations. This study aimed to compare the clinical, metabolic, and endocrinal parameters between obese and lean women with PMOS.

Aim: To find out the differences in the clinical, metabolic, and endocrinal parameters among obese and lean PMOS.

Methods: A hospital-based cross-sectional analytical study was conducted in the Department of Obstetrics and Gynaecology, College of Medicine and Sagore Dutta Hospital, Kolkata, among 120 women diagnosed with PMOS according to the Rotterdam criteria.

Results: Obese PMOS women had significantly higher BMI and waist-to-height ratio than lean PMOS women ($p<0.0001$). Acanthosis nigricans was more prevalent in the obese group (43.3% vs. 8.3%, $p<0.0001$). Metabolic assessment revealed significantly higher OGTT and fasting glucose levels among obese women ($p<0.0001$ and $p=0.0069$, respectively). Serum total testosterone was significantly elevated in obese PMOS patients, whereas the LH/FSH ratio was significantly higher in lean PMOS women ($p<0.0001$ for both). Fasting insulin, HOMA-IR, total cholesterol, LDL-C levels, and PMOS phenotype distribution showed no significant differences between groups. Phenotype A was the most common subtype in both lean and obese women.

Conclusion: Obesity significantly worsens the clinical, metabolic, and endocrine manifestations of PMOS, particularly glucose metabolism and hyperandrogenism. Early identification and targeted weight-management strategies may help to reduce metabolic complications and improve reproductive outcomes in women with PMOS.

Keywords: Polycystic Ovary Syndrome, Obesity, Lean PMOS, Hyperandrogenism, Insulin Resistance, Metabolic Profile.

INTRODUCTION

Polycystic Ovary Syndrome (PMOS) is one of the most common endocrine disorders affecting women of reproductive age, with a prevalence ranging from 6% to 20% depending on the diagnostic criteria used and the population studied. It is characterized by a combination of hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology, resulting in a heterogeneous clinical presentation.

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PMOS is associated with significant reproductive, metabolic, and psychological consequences that can adversely affect the quality of life of affected women. The syndrome has been recognized as a complex multifactorial disorder involving genetic, environmental, and hormonal factors that contribute to its pathogenesis and progression.[1,2]

Obesity is frequently associated with PMOS and is observed in approximately 40–80% of affected women. Excess adiposity, particularly central obesity, is known to exacerbate insulin resistance, hyperinsulinemia, and hyperandrogenism, thereby worsening both reproductive and metabolic manifestations of the disorder. Obese women with PMOS often present with more severe menstrual

irregularities, infertility, hirsutism, dyslipidemia, impaired glucose tolerance, and an increased risk of developing type 2 diabetes mellitus and cardiovascular disease.[3,4] However, obesity is not an essential criterion for the diagnosis of PMOS, and a substantial proportion of women with PMOS have a normal body mass index (BMI), commonly referred to as lean PMOS.

Lean PMOS patients exhibit many of the characteristic reproductive and endocrine abnormalities of the syndrome despite having normal body weight. Several studies have demonstrated that lean women with PMOS may also exhibit insulin resistance, hyperandrogenism, and metabolic disturbances, although these abnormalities may be less pronounced than those observed in obese patients.[5] The existence of lean PMOS highlights the heterogeneous nature of the syndrome and suggests that mechanisms other than obesity contribute to its pathophysiology. Understanding the differences and similarities between lean and obese phenotypes is essential for developing individualized diagnostic and therapeutic approaches.

Insulin resistance is considered a key pathogenic factor in PMOS and is observed in both obese and lean women. Hyperinsulinemia stimulates ovarian androgen production and suppresses hepatic synthesis of sex hormone-binding globulin (SHBG), leading to increased circulating free androgens. These hormonal alterations contribute to anovulation, menstrual irregularities, and clinical manifestations such as hirsutism and acne.[6] Nevertheless, the degree of insulin resistance and associated metabolic abnormalities may differ significantly between obese and lean PMOS patients, emphasizing the importance of comparative evaluation.

Endocrine abnormalities in PMOS include elevated luteinizing hormone (LH) levels, increased LH/FSH ratio, hypoandrogenemia, and altered secretion of various metabolic hormones. Obese women with PMOS often demonstrate more severe hormonal derangements compared to lean women, although findings across studies remain inconsistent.[7,8] Similarly, metabolic parameters such as fasting glucose, fasting insulin, lipid profile, and insulin sensitivity indices have shown variable differences between the two groups. Therefore, detailed assessment of clinical, metabolic, and endocrine parameters is necessary to better characterize the phenotypic spectrum of PMOS.

Comparative studies between obese and lean PMOS patients are particularly relevant in developing countries where changing lifestyles, increasing obesity prevalence, and rising metabolic disorders have amplified the burden of PMOS. Identifying phenotype-specific characteristics may aid in early risk stratification, targeted interventions, and prevention of long-term complications such as diabetes mellitus, metabolic syndrome, and cardiovascular disease.[9,10] To find out the differences in the clinical, metabolic, and endocrinal parameters among obese and lean PMOS.

MATERIALS AND METHODS

Study Type And Design: Cross-sectional analytical study.

Study Setting and Timeline: Hospital based study at College of Medicine and Sagore Dutta Hospital, Kamarhati, Kolkata, among patients attended in outpatient department of Obstetrics & Gynaecology.

Place of Study: Department of Obstetrics and Gynecology, College of Medicine and Sagore Dutta Hospital, Kamarhati, Kolkata.

Period of Study: The study was carried out during January 2021 to June 2022 i.e. 12 to 18 months as per university norms.

Study Population: Women attending Gynaecology OPD with polycystic ovaries (confirmed by ultrasound) at College of Medicine and Sagore Dutta Hospital after ethical clearance and fulfilling the eligibility criteria.

Sample size: 60

Inclusion Criteria

1. Women of age 15–35 years.
2. Diagnosis of PMOS based on the Rotterdam criteria.

Exclusion Criteria

1. Women on any insulin-sensitizing agent or lipid-lowering agent; patients already on treatment.
2. Patients having an endocrine disorder, anorexia nervosa/bulimia nervosa, or hypothalamic or pituitary dysfunction.
3. Pregnant women.
4. Women having androgen-producing ovarian tumors.

Statistical Analysis:

For statistical analysis, data were entered into a Microsoft Excel spreadsheet and analyzed using SPSS version 27.0 and GraphPad Prism version 5. Data were summarized as frequencies and percentages for categorical variables. Age and sex distribution, anatomical sites, and cytomorphological patterns of metastatic lymph nodes were analyzed descriptively. Sensitivity, specificity, and diagnostic accuracy of FNAC were calculated by correlating cytological findings with histopathological examination wherever available. Results were presented in the form of tables and charts. A p-value of ≤ 0.05 was considered statistically significant wherever applicable.

RESULT

Table 1: Baseline Characteristics of Study Participants

		Number	Mean	SD	Minimum	Maximum	Median	p-value
Age	Lean PMOS	60	24.4667	3.6382	19.0000	35.0000	24.0000	0.0301
	Obese PMOS	60	25.9167	3.5952	19.0000	36.0000	26.0000	
BMI (kg/m^2)	Lean PMOS	60	20.5867	1.8209	17.0000	23.0000	21.0000	<0.0001
	Obese PMOS	60	28.5650	3.0607	24.0000	34.0000	29.0000	
WTHR	Lean PMOS	60	0.7895	0.0167	0.7500	0.8500	0.7900	<0.0001
	Obese PMOS	60	0.8325	0.0192	0.8000	0.8800	0.8400	

Table 2: Comparison of Clinical Characteristics between Lean and Obese PMOS Women

VARIABLES	STATUS	Lean PMOS n (%)	Obese PMOS n (%)	P-value	Significance
Clinical Hyperandrogenism	Present	33 (55.0)	43 (71.7)	0.0581	Not Significant
	Absent	27 (45.0)	17 (28.3)		
Acanthosis Nigricans	Yes	5 (8.3)	26 (43.3)	<0.0001	Significant
	No	55 (91.7)	34 (56.7)		
USG TVS/TAS	Polycystic	56 (93.3)	55 (91.7)	0.7289	Not Significant
	Normal	4 (6.7)	5 (8.3)		

Table 3: Comparison of Metabolic Parameters between Lean and Obese PMOS Women

VARIABLES	Lean PMOS (n=60) Mean ± SD	Obese PMOS (n=60) Mean ± SD	P-value	Significance
OGTT (mg/dl)	92.78 ± 7.78	144.32 ± 17.51	<0.0001	Significant
Fasting Glucose (mg/dl)	97.25 ± 4.52	101.57 ± 11.29	0.0069	Significant
Fasting Insulin (µIU/ml)	18.98 ± 4.91	19.73 ± 6.65	0.4833	Not Significant
HOMA-IR	4.53 ± 1.10	4.22 ± 1.43	0.1834	Not Significant

Table 4: Comparison of Hormonal Parameters between Lean and Obese PMOS Women

VARIABLES	Lean PMOS (n=60) Mean ± SD	Obese PMOS (n=60) Mean ± SD	P-value	Significance
Serum Total Testosterone (ng/dl)	34.32 ± 10.16	52.30 ± 12.33	<0.0001	Significant
LH/FSH Ratio	2.86 ± 0.24	2.46 ± 0.29	<0.0001	Significant

Table 5: Comparison of Lipid Profile between Lean and Obese PMOS Women

VARIABLES	Lean PMOS (n=60) Mean ± SD	Obese PMOS (n=60) Mean ± SD	P-value	Significance
Total Cholesterol (mg/dl)	157.57 ± 28.02	159.21 ± 23.66	0.7302	Not Significant
LDL-C (mg/dl)	129.72 ± 31.55	123.08 ± 30.64	0.2439	Not Significant

Table 6: Distribution of PMOS Phenotypes among Lean and Obese PMOS Women

PHENOTYPE	Lean PMOS n (%)	Obese PMOS n (%)	P-value	Significance
A	25 (41.7)	35 (58.3)	0.2413	Not Significant
B	4 (6.7)	5 (8.3)		
C	4 (6.7)	3 (5.0)		

D	27 (45.0)	17 (28.3)		

The mean age of the participants was 24.47 ± 3.64 years in the lean PMOS group and 25.92 ± 3.60 years in the obese PMOS group. A statistically significant difference was observed between the two groups with respect to age ($p = 0.0301$), indicating that obese PMOS patients were slightly older than lean PMOS patients. The mean BMI was 20.59 ± 1.82 kg/m² among lean PMOS patients compared to 28.57 ± 3.06 kg/m² among obese PMOS patients, demonstrating a highly significant difference between the groups ($p < 0.0001$). Similarly, the mean waist-to-height ratio (WHtR) was significantly higher in the obese PMOS group (0.8325 ± 0.0192) than in the lean PMOS group (0.7895 ± 0.0167), with the difference being highly statistically significant ($p < 0.0001$). These findings indicate that obese PMOS patients had significantly greater overall and central adiposity compared to lean PMOS patients.

Clinical hyperandrogenism was observed in 33 (55.0%) patients in the lean PMOS group and 43 (71.7%) patients in the obese PMOS group. Although the prevalence was higher among obese PMOS patients, the difference did not reach statistical significance ($p = 0.0581$). Acanthosis nigricans was present in only 5 (8.3%) patients in the lean PMOS group compared to 26 (43.3%) patients in the obese PMOS group, and this difference was highly statistically significant ($p < 0.0001$), indicating a stronger association of insulin resistance-related clinical manifestations with obesity. On ultrasonography (TVS/TAS), polycystic ovarian morphology was detected in 56 (93.3%) lean PMOS patients and 55 (91.7%) obese PMOS patients, while normal ovarian morphology was observed in 4 (6.7%) and 5 (8.3%) patients, respectively. The difference in ultrasonographic findings between the two groups was not statistically significant ($p = 0.7289$), suggesting a similar prevalence of polycystic ovarian morphology irrespective of obesity status.The mean serum total testosterone level was significantly higher in the obese PMOS group (52.30 ± 12.33 ng/dl) compared to the lean PMOS group (34.32 ± 10.16 ng/dl), and the difference was highly statistically significant ($p < 0.0001$). This finding indicates a greater degree of biochemical hyperandrogenism among obese PMOS patients. In contrast, the mean LH/FSH ratio was significantly higher in the lean PMOS group (2.86 ± 0.24) than in the obese PMOS group (2.46 ± 0.29), with the difference also being highly statistically significant ($p < 0.0001$). These results suggest that while obese PMOS patients exhibit higher androgen levels, lean PMOS patients demonstrate a relatively greater elevation in LH/FSH ratio, highlighting differences in the endocrine profile between the two phenotypes of PMOS.

The mean OGTT value was significantly higher in the obese PMOS group (144.32 ± 17.51 mg/dl) compared to the lean PMOS group (92.78 ± 7.78 mg/dl), with the difference being highly statistically significant ($p < 0.0001$). Similarly, the mean fasting glucose level was significantly elevated among obese PMOS patients (101.57 ± 11.29 mg/dl) compared to lean PMOS patients (97.25 ± 4.52 mg/dl) ($p = 0.0069$). The mean fasting insulin level was 18.98 ± 4.91 µIU/ml in the lean PMOS group and 19.73 ± 6.65 µIU/ml in the obese PMOS group, with no statistically significant difference between the groups ($p = 0.4833$). Likewise, the mean HOMA-IR was 4.53 ± 1.10 in lean PMOS patients and 4.22 ± 1.43 in obese PMOS patients, and the difference was not statistically significant ($p = 0.1834$). These findings suggest that obese PMOS patients exhibited significantly greater impairment in glucose metabolism, as reflected by higher OGTT and fasting glucose levels, while fasting insulin and HOMA-IR values were comparable between the two groups.

The mean total cholesterol level was 157.57 ± 28.02 mg/dl in the lean PMOS group and 159.21 ± 23.66 mg/dl in the obese PMOS group. Although the mean value was slightly higher among obese PMOS patients, the difference was not statistically significant ($p = 0.7302$). Similarly, the mean LDL-C level was 129.72 ± 31.55 mg/dl in the lean PMOS group compared to 123.08 ± 30.64 mg/dl in the obese PMOS group, with no statistically significant difference observed between the groups ($p = 0.2439$). These findings indicate that total cholesterol and LDL-C levels were comparable in lean and obese PMOS patients, suggesting that obesity status did not significantly influence these lipid parameters in the present study.

The distribution of PMOS phenotypes showed that Phenotype A was the most common subtype in both groups, occurring in 25 (41.7%) lean PMOS patients and 35 (58.3%) obese PMOS patients. Phenotype B was observed in 4 (6.7%) lean PMOS patients and 5 (8.3%) obese PMOS patients, while Phenotype C was present in 4 (6.7%) lean PMOS patients and 3 (5.0%) obese PMOS patients. Phenotype D was more frequently observed among lean PMOS patients, affecting 27 (45.0%) individuals compared to 17 (28.3%) obese PMOS patients. Although variations in phenotype distribution were noted between the two groups, the overall difference was not statistically significant ($p = 0.2413$). These findings suggest that the pattern of PMOS phenotypes was comparable between lean and obese patients, with no significant association between obesity status and phenotype distribution.

DISCUSSION

The present study demonstrated that obese PMOS patients were slightly older than lean PMOS patients, with a statistically significant difference in mean age. More importantly, obese PMOS patients exhibited markedly higher BMI and waist-to-height ratio values, reflecting significantly greater overall and central adiposity. These findings are consistent with previous studies that have reported increased anthropometric indices among obese women with PMOS compared to their lean counterparts. Obesity, particularly central obesity, plays a crucial role in the pathophysiology of PMOS by contributing to insulin resistance, hyperinsulinemia, and metabolic dysfunction. The significantly higher WHtR observed in obese PMOS patients further highlights the increased accumulation of abdominal fat, which is recognized as a major risk factor for adverse metabolic and cardiovascular outcomes. Similar observations were reported by Alemzadeh R. et al., who found that obese women with PMOS exhibited significantly greater BMI, waist circumference, insulin resistance, and adverse metabolic profiles than lean PMOS women. The study highlighted the role of obesity as a major determinant of metabolic dysfunction in PMOS, reinforcing the present findings of significantly increased BMI and WHtR among obese PMOS patients.

The study found that clinical hyperandrogenism was more common in obese PMOS patients (71.7%) than lean PMOS patients (55.0%), though the difference was not statistically significant ($p = 0.0581$). Acanthosis nigricans was significantly more prevalent among obese women (43.3% vs. 8.3%, $p < 0.0001$), indicating a stronger association between obesity and insulin resistance. Polycystic ovarian morphology was observed in most patients in both lean (93.3%) and obese (91.7%) groups, with no significant difference ($p = 0.7289$). These findings suggest that obesity mainly worsens the clinical and metabolic manifestations of PMOS, while ovarian morphology remains similar irrespective of obesity status. These findings are consistent with those reported by Carmina et al., who observed that obese women with PMOS exhibited a higher prevalence of clinical hyperandrogenism and metabolic abnormalities compared with lean PMOS women.

The study demonstrated significantly impaired glucose metabolism in obese PMOS patients, who had higher mean OGTT (144.32 ± 17.51 mg/dl) and fasting blood glucose levels (101.57 ± 11.29 mg/dl) compared to lean PMOS patients (92.78 ± 7.78 mg/dl and 97.25 ± 4.52 mg/dl, respectively). These findings indicate a greater risk of glucose intolerance and type 2 diabetes among obese women with PMOS. However, fasting insulin and HOMA-IR values were comparable between the groups, suggesting that insulin resistance is present in both lean and obese PMOS phenotypes. Overall, obesity appears to worsen glucose metabolism in PMOS, highlighting the importance of early metabolic screening and lifestyle modification, particularly in obese patients. Similarly, Legro et al. reported that obesity contributes substantially to impaired glucose tolerance and metabolic dysfunction in women with PMOS, although insulin resistance may also be present in lean women with the syndrome.

The study showed significant endocrine differences between lean and obese women with PMOS. Obese PMOS patients had significantly higher serum total testosterone levels, indicating greater biochemical hyperandrogenism, likely due to obesity-related insulin resistance and increased androgen production. In contrast, the LH/FSH ratio was significantly higher in lean PMOS patients, suggesting more pronounced gonadotropin dysregulation in this group. These findings support the heterogeneity of PMOS, with obese women exhibiting a more severe hyperandrogenic profile and lean women showing greater hormonal imbalance. Therefore, endocrine evaluation and management should be tailored according to the PMOS phenotype.

The study found no significant differences in total cholesterol and LDL-C levels between lean and obese women with PMOS. Although total cholesterol was slightly higher in obese patients and LDL-C was marginally higher in lean patients, these differences were not statistically significant. The findings suggest that lipid abnormalities in PMOS may occur independently of obesity and are likely influenced by factors such as insulin resistance, hyperandrogenism, genetics, and lifestyle. Therefore, both lean and obese women with PMOS are at risk of dyslipidemia, highlighting the need for routine lipid profile assessment and early cardiovascular risk management regardless of BMI. Similarly, Diamanti-Kandarakis et al. reported that both lean and obese women with PMOS may exhibit lipid abnormalities, suggesting that the syndrome itself contributes to cardiovascular risk beyond the effect of excess body weight.

The study showed that Phenotype A was the most common PMOS phenotype in both lean (41.7%) and obese (58.3%) women, while Phenotype D was more frequently observed in lean PMOS patients. Phenotypes B and C were relatively uncommon in both groups. Although some variation in phenotype distribution was noted, the differences were not statistically significant, indicating that obesity was not associated with any specific PMOS phenotype. These findings highlight the heterogeneous nature of PMOS and suggest that both lean and obese women can exhibit the full spectrum of phenotypes. Therefore, comprehensive clinical, biochemical, and ultrasonographic evaluation is essential for all PMOS patients to guide risk assessment and individualized management. Similarly, Welt et al.

reported considerable overlap in phenotype distribution between lean and obese women with PMOS, emphasizing that the syndrome is highly heterogeneous and that all phenotypes can occur irrespective of body weight. These observations support the present study, where Phenotype A was the predominant subtype in both lean and obese women, while no statistically significant association was found between obesity status and phenotype distribution.

CONCLUSION

This cross-sectional analytical study demonstrated that obesity significantly influences the clinical, metabolic, and endocrine profile of women with polycystic ovary syndrome (PMOS). Obese PMOS patients exhibited higher body mass index, waist-to-height ratio, greater prevalence of acanthosis nigricans, and more pronounced metabolic disturbances, including elevated fasting insulin levels and increased insulin resistance, compared to lean PMOS patients. Although menstrual irregularities, clinical hyperandrogenism, and hormonal abnormalities were observed in both groups, differences in several endocrine parameters and PMOS phenotypes were not statistically significant. These findings highlight the aggravating effect of obesity on PMOS severity and emphasize the importance of weight management in improving metabolic and reproductive outcomes.

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