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# Prostate Specific Antigen (PSA) Level in Lean versus Obese Polycystic Ovarian Syndrome (PCOS)

**Md. Ashraf Uddin[1*], Mohammad Syedul Islam[2], Quazi Mamtaz Uddin Ahmed[3], Imtiaz Ahmed[4], Md. Mahadi Hassan Bhuyan[5], Ishtiak Khan[6], Md. Ariful Islam[7], Ruhus Safa Noor[8], Fahmida Faizha Shama[9]**

[1]Medical Officer, Directorate General of Health Services, Mohakhali, Dhaka, Bangladesh.
[2]Associate Professor, Department of Internal Medicine, Bangladesh Medical University, Dhaka, Bangladesh.
[3]Professor, Department of Internal Medicine, Bangladesh Medical University, Dhaka, Bangladesh.
[4]Assistant Professor, Department of Internal Medicine, Bangladesh Medical University, Dhaka, Bangladesh.
[5]Resident (Phase B), Department of Internal Medicine, Bangladesh Medical University, Dhaka, Bangladesh.
[6]Assistant Surgeon, Department of Emergency, Lalmonirhat 250 Bed Sadar Hospital, Lalmonirhat, Bangladesh.
[7]Associate Professor (cc) & Head, Department of ENT & Head Neck Surgery, Dhaka Community Medical Collage, Boro Mogbazar, Wireless, Dhaka, Bangladesh.
[8]Medical Officer, Department of Dermatology, Mugda Medical College, Mugda, Dhaka, Bangladesh.
[9]MPhil (on course), National Institute of Preventive and Social Medicine, Mohakhali, Dhaka, Bangladesh.

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## ABSTRACT

**Background:** Polycystic ovarian syndrome is a metabolic and hormonal disorder marked by irregular cycles, hyperandrogenism, and insulin resistance. While both lean and obese women show adverse metabolic profiles, obesity increases diabetes risk. PSA is elevated in PCOS, stable, and easily measured, making it a potential diagnostic marker for PCOS. This study compared clinical, metabolic, hormonal factors, and PSA in lean versus obese PCOS women in Bangladesh. **Methods:** This cross-sectional research conducted at BMU, Dhaka, involved 63 women diagnosed with PCOS (obese ≥23 kg/m², n=33; lean <23 kg/m², n=30). Clinical, biochemical, and hormonal information, including PSA, was gathered. Statistical evaluation employed conventional tests ($p<0.05$). Women with additional endocrine disorders, pregnancy, breastfeeding, or recent treatment were not included. Approval for ethics and consent from participants were secured. **Results:** In a study of 63 women with PCOS, the obese group exhibited significantly higher median PSA levels (0.053 vs 0.051 ng/ml, $p=0.008$), systolic blood pressure (112.4 ± 10.8 vs 101.3 ± 6.3 mmHg, $p<0.001$), and diastolic blood pressure (75.3 ± 8.2 vs 69.2 ± 7.2 mmHg, $p=0.003$), Ferriman–Gallwey scores (median 8 vs 2, $p=0.043$), HbA1C (5.81 ± 0.21 vs 5.57 ± 0.22, $p<0.001$), triglycerides (140.7 ± 89.3 vs 86.3 ± 27.3 mg/dl, $p<0.001$), as well as testosterone levels (47.5 ± 41.6 vs 31.3 ± 33.3 ng/dl, $p=0.03$). **Conclusion:** Women with obesity and PCOS exhibited elevated levels of PSA, testosterone, blood pressure, hirsutism, acne, acanthosis nigricans, HbA1C, and triglycerides compared to lean women, whereas other hormone levels were comparable. Obesity showed a strong association with clinical characteristics related to hyperandrogenism and insulin resistance..



## INTRODUCTION:

Polycystic ovarian syndrome (PCOS) is a complex metabolic and endocrine disorders consisting of clinical symptoms like chronic anovulation or oligo-ovulation, menstrual irregularities, symptoms of hyperandrogenism like acne, hirsutism, obesity and hyperinsulinemia [1]. The occurrence of this disorder worldwide typically ranges from 5% to 15% [2]. In 2012, The World Health Organization (WHO) estimates that about 116 million women (3.4%) globally were impacted by PCOS [1]. PCOS is still

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poorly understood regarding consistent diagnosis and treatment criteria [3].

PCOS manifestations may begin in adolescence but are often diagnosed in adulthood [4]. Adolescent PCOS diagnosis involves a thorough assessment of symptoms like hirsutism, acne, irregular menstrual cycles, and elevated androgen levels two years post-menarche [5]. Apart from the clinical manifestation like hirsutism, hyperandrogenism may manifest biochemically as increased levels of free testosterone, total testosterone, and other circulating androgenic steroids and metabolites [2]. Ultrasound morphology of polycystic ovary without signs of hyperandrogenism or menstrual irregularities should not be utilized for diagnosing PCOS in adolescents [5]. By utilizing biochemical and/or clinical evidence, epidemiological research estimates 6.5% to 8% prevalence among reproductive-age women, where ultrasound studies showing a 20% or higher prevalence [6].

Obesity is linked to worsened metabolic and ovulatory issues associated with PCOS [7]. Obesity is strongly linked to PCOS, with 38%-88% of women with PCOS classified as obese or overweight. A meta-analysis studies indicates that women with obesity have a 2.77 odds ratio for developing PCOS compared to non- obese women [8]. PCOS is linked to alterations in lipid profiles and an increase in small, dense LDL particles [9]. In Northern Finland Birth Cohort (NFBC) links correlation between PCOS traits and BMI and, as well as childhood adiposity rebound leading to adult PCOS [10]. On the other side, losing weight has been shown to restore ovulation and enhance hyperandrogenism [7].

Androgens and progestins upregulate the PSA gene in female tissues. Increased serum and urine PSA levels have been observed in hirsute women and individuals with PCOS [2]. PSA level serves as a diagnostic marker for PCOS, demonstrating relatively high specificity (80%) and sensitivity (70%) [11]. Serum PSA levels are stable across the menstrual cycle, exhibiting minimal intracyclic variations [12]. Unlike ultrasound ovarian morphology, PSA is not operator-dependent and is widely accessible. Therefore, PSA may serve as a diagnostic test for PCOS [13]. PCOS can affect women of all weights, not just those who are overweight [14]. Both lean and obese women with PCOS have poorer metabolic profiles and similar visceral fat compared to controls, indicating that nonobese PCOS carries metabolic risks comparable to obese PCOS [15]. Obese women had increased risk for diabetes and other morbidities compared to lean women [16]. Research on PSA levels in women across BMI categories is limited, but some studies suggest PSA is higher in obese and hirsute women with PCOS [17,18].

PSA is a simple, operator-independent test with potential as a diagnostic marker for PCOS. Studies comparing PSA between lean and obese PCOS women are limited, but analyzing PSA may provide insights into hyperandrogenism and related clinical, metabolic, and hormonal differences. This study aimed to compare these parameters and PSA levels between lean and obese PCOS patients in Bangladesh.

## MATERIALS AND METHODS

### Study Design and Setting

This cross-sectional analytical study was conducted in the Department of Internal Medicine, Bangladesh Medical University (BMU), Dhaka, Bangladesh, from April 2024 to September 2025. The primary aim was to compare serum prostate-specific antigen (PSA) levels between obese and lean women diagnosed with polycystic ovary syndrome (PCOS) and to explore its associations with clinical, biochemical, and hormonal parameters.

### Study Population

A total of 63 women with PCOS were enrolled. Participants were classified into two groups based on body mass index (BMI):

- Obese group: BMI ≥ 23 kg/m² (n=33)
- Lean group: BMI < 23 kg/m² (n=30)

PCOS was diagnosed according to the Rotterdam criteria, which requires at least two of the following: oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovaries on ultrasound.

### Inclusion and Exclusion Criteria

**Inclusion criteria:**

- Women aged 18–35 years diagnosed with PCOS
- Willingness to participate and provide informed consent

**Exclusion criteria:**

- History of endocrine disorders other than PCOS (e.g., thyroid dysfunction, Cushing's syndrome)
- Current pregnancy or lactation
- Use of hormonal medications, insulin sensitizers, or lipid-lowering drugs within three months prior to the study

### Data Collection:

**Clinical Assessment:**

Sociodemographic data, including age, marital status, occupation, education, and self-perceived socioeconomic status, were collected using a structured questionnaire. Clinical evaluation included:

- Anthropometry: Height, weight, and BMI calculation
- Blood pressure: Systolic and diastolic readings

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- Modified Ferriman Gallway (FGS) score: Assessment of hirsutism

**Biochemical and Hormonal Assessment:**
Blood samples were collected after overnight fasting. The following parameters were measured:

- Biochemical: HbA1C, fasting lipid profile (cholesterol, triglycerides, HDL, LDL)
- Hormonal: Serum PSA, testosterone, progesterone, FSH, LH, LH:FSH ratio, TSH, and prolactin Serum PSA was measured using a standardized immunoassay.

**Statistical Analysis:**
All data were analyzed using SPSS version 26.0. Continuous variables were summarized as mean ± standard deviation (SD) for normally distributed data, and as median with interquartile range (IQR) for skewed distributions, while categorical variables were expressed as frequencies and percentages. Comparisons between obese and lean PCOS groups were performed using the independent t-test for normally distributed continuous variables and the Mann–Whitney U test for non-normally distributed variables. Associations between categorical variables were assessed with the chi-square test. To explore relationships between serum PSA levels and clinical, biochemical, and hormonal parameters, Pearson correlation was applied for normally distributed variables and Spearman correlation for non-normally distributed or ordinal variables. A p-value $< 0.05$ was considered statistically significant in all analyses.

**Ethical Considerations:**
The study protocol was approved by the Institutional Review Board of BMU. Written informed consent was obtained from all participants prior to enrollment, and confidentiality of patient information was maintained throughout the study.

## RESULTS:
A total of 63 women diagnosed with polycystic ovary syndrome (PCOS) were included in the study, of whom 33 (52.4%) were classified as obese and 30 (47.6%) as lean based on body mass index (BMI) criteria by WHO for Asian Population.

**Sociodemographic Characteristics:**
Table I shows in the present study, the patient ranged from 18 years to 32 years. The mean age was found slightly higher in obese PCOS patients (24.09 years) than lean PCOS patients (23 years). Among the respondents, 87.9% obese PCOS patients were married and 76.7% lean PCOS patients were married. Education level up to SSC and below were found more in obese PCOS patients. HSC and honors above were found higher proportion in lean PCOS patients, 26.7% and 46.7% respectively. Majority of the patients were housewife and obese patients had more housewife than lean patients. Self-perceived economic status was middle class among majority of the patients and proportion found more in lean PCOS patient group (90%) than obese patients (87.9%). No statistically significant differences were observed in sociodemographic variables between the two groups.

**Table I. Sociodemographic Characteristics of Obese and Lean PCOS Patients (n=63)**

| Variable | Obese (n=33) | Lean (n=30) | p-value |
|---|---|---|---|
| **Age (years)** | | | 0.25 |
| Mean ± SD | 24.09 ± 3.21 | 23.10 ± 3.59 | |
| Range | (18–30) | (18–32) | |
| **Marital status, n (%)** | | | 0.24 |
| Married | 29 (87.9) | 23 (76.7) | |
| Unmarried | 4 (12.1) | 7 (23.3) | |
| **Occupation, n (%)** | | | 0.50 |
| Housewife | 25 (75.8) | 22 (73.3) | |
| Student | 5 (15.2) | 7 (23.3) | |
| Service | 3 (9.1) | 1 (3.3) | |
| **Education, n (%)** | | | 0.56 |
| SSC and below | 13 (39.4) | 8 (26.7) | |
| HSC | 7 (21.2) | 8 (26.7) | |
| Honors and above | 13 (39.4) | 14 (46.7) | |
| **Socioeconomic status, n (%)** | | | 0.94 |
| Lower middle | 3 (9.1) | 2 (6.7) | |
| Middle | 29 (87.9) | 27 (90.0) | |
| Upper middle | 1 (3.0) | 1 (3.3) | |

**Note: Independent t-test and Chi-square test applied; p<0.05 considered statistically significant**

**Serum PSA Levels:**
Figure 1 presents Serum PSA levels were evaluated using a box-and-whisker plot. The median PSA for obese women was 0.05370 ng/ml (IQR 0.0049, range 0.0400–0.0588 ng/ml), whereas for lean women it was 0.05175 ng/ml (IQR 0.0062, range 0.0400–0.0568 ng/ml). Whiskers represent the minimum and maximum values, the box represents the interquartile range, and the line inside the box indicates the median; asterisks represent extreme values.

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Table II shows comparison of mean PSA levels between the groups showed that obese women had significantly higher PSA than lean women (0.053 ± 0.004 ng/ml vs 0.050 ± 0.004 ng/ml, p=0.008).

**Table 2. Comparison of Serum PSA Levels between Obese and Lean PCOS Patients (n=63)**

| Parameter | Obese (n=33) | Lean (n=30) | p-value |
|---|---|---|---|
| PSA (ng/ml) * | 0.053 ± 0.004 | 0.050 ± 0.004 | 0.008[t] |

*Arithmetic mean ± SD; Independent t-test applied; p<0.05 considered significant

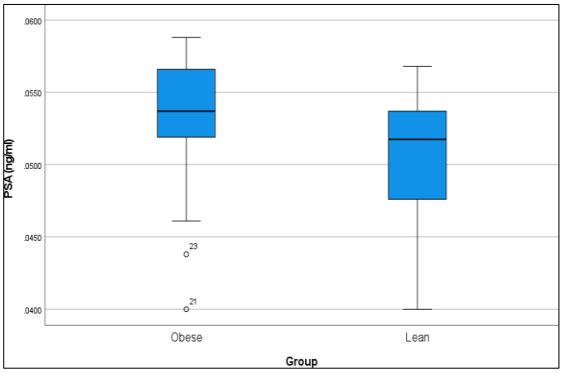


**Figure 1: Serum PSA level between the group**

In figure 1 Whiskers box and plot graph showed the level of PSA in obese and lean patients of PCOS. Box represents the range from the first to third quartile. Line inside the box showed the median of the value. Whiskers represent the range between maximum and minimum. Asters represents extreme values.

For obese group, median value of PSA 0.05370 with IQR 0.0049 ranging from 0.0400 to 0.0588 ng/ml. For lean group, median value of PSA 0.051750 with IQR 0.0062 ranging from 0.0400 to 0.0568 ng/ml.

**Clinical Characteristics:**

Table III presents obese women exhibited significantly higher systolic and diastolic blood pressure compared to lean women (112.38 ± 10.83 vs 101.31 ± 6.32 mmHg, p<0.001; and 75.30 ± 8.19 vs 69.17 ± 7.20 mmHg, p=0.003, respectively).

Clinical hyperandrogenism, assessed using the Modified Ferriman–Gallwey (FG) score, was significantly more pronounced among obese participants (median 8, IQR 12) compared to lean participants (median 2, IQR 11; p=0.043).

Furthermore, the prevalence of acne vulgaris and acanthosis nigricans was significantly higher in obese women than in lean women (57.6% vs 30.0%, p=0.03; and 60.6% vs 6.7%, p<0.001, respectively).

**Table III. Comparison of Clinical Characteristics between Obese and Lean PCOS Patients (n=63)**

| Variable | Obese (n=33) | Lean (n=30) | p-value |
|---|---|---|---|
| Systolic BP (mmHg) | 112.38 ± 10.83 | 101.31 ± 6.32 | <0.001 |
| Diastolic BP (mmHg) | 75.30 ± 8.19 | 69.17 ± 7.20 | 0.003 |
| Ferriman–Gallwey Score | 8 (IQR 12) | 2 (IQR 11) | 0.043 |
| Acne vulgaris, n (%) | 19 (57.6) | 9 (30.0) | 0.03 |
| Acanthosis nigricans, n (%) | 20 (60.6) | 2 (6.7) | <0.001 |

**Metabolic Profile:**

Table IV shows Obese women demonstrated significantly higher levels of glycated hemoglobin (HbA1C) compared to lean women (5.81 ± 0.21 vs 5.57 ± 0.22, p<0.001).

Similarly, serum triglyceride levels were significantly elevated in the obese group (140.72 ± 89.29 mg/dl) compared to the lean group (86.34 ± 27.25 mg/dl, p<0.001).

However, no statistically significant differences were observed between the two groups in terms of total cholesterol, high-density lipoprotein (HDL), and low-density lipoprotein (LDL) levels (p>0.05 for all).

**Table IV. Comparison of Metabolic Parameters between Obese and Lean PCOS Patients (n=63)**

| Variable | Obese (n=33) | Lean (n=30) | p-value |
|---|---|---|---|
| HbA1C (%) | 5.81 ± 0.21 | 5.57 ± 0.22 | <0.001 |
| Triglyceride (mg/dl) | 140.72 ± 89.29 | 86.34 ± 27.25 | <0.001 |
| Total Cholesterol (mg/dl) | 178.14 ± 41.92 | 166.33 ± 27.05 | 0.19 |
| HDL (mg/dl) | 43.03 ± 10.19 | 46.10 ± 6.68 | 0.16 |
| LDL (mg/dl) | 114.68 ± 31.75 | 102.28 ± 24.94 | 0.09 |

**Hormonal Profile:**

Table V shows Serum testosterone levels were significantly higher among obese women compared to lean women (47.45 ± 41.61 vs 31.33 ± 33.26 ng/dl, p=0.03), indicating greater biochemical hyperandrogenism in the obese phenotype.

In contrast, no statistically significant differences were observed in other hormonal parameters, including progesterone, thyroid-stimulating hormone (TSH), luteinizing hormone (LH), follicle-stimulating hormone (FSH), LH:FSH ratio, and prolactin levels between the two groups (p>0.05 for all).

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**Table V. Comparison of Hormonal Parameters between Obese and Lean PCOS Patients (n=63)**

| Variable | Obese (n=33) | Lean (n=30) | p-value |
|---|---|---|---|
| Testosterone (ng/dl) | 47.45 ± 41.61 | 31.33 ± 33.26 | 0.03 |
| Progesterone (ng/ml) | 0.50 ± 0.11 | 0.51 ± 0.11 | 0.77 |
| TSH (µIU/ml) | 2.45 ± 1.68 | 2.25 ± 1.53 | 0.58 |
| LH (µIU/ml) | 7.06 ± 8.05 | 5.75 ± 6.77 | 0.32 |
| FSH (µIU/ml) | 5.28 ± 2.61 | 5.77 ± 1.60 | 0.35 |
| LH: FSH Ratio | 1.33 ± 1.16 | 0.99 ± 1.31 | 0.14 |
| Prolactin (ng/ml) | 10.82 ± 7.57 | 11.44 ± 7.00 | 0.68 |

**Risk Estimation of Clinical Features:**
Table VI presents obesity was associated with a significantly increased likelihood of clinical manifestations of hyperandrogenism. The odds of developing acne vulgaris were more than three times higher among obese women (OR=3.17, 95% CI: 1.12–8.98).

Notably, the odds of acanthosis nigricans were markedly elevated in the obese group (OR=21.53, 95% CI: 4.36–106.19), indicating a strong association between obesity and insulin resistance–related dermatological manifestations.

**Table VI. Odds Ratio for Clinical Features among Obese vs Lean PCOS Patients**

| Feature | Odds Ratio (OR) | 95% CI |
|---|---|---|
| Acne vulgaris | 3.17 | 1.12–8.98 |
| Acanthosis nigricans | 21.53 | 4.36–106.19 |

## DISCUSSION:
In this study, the average age was marginally greater in obese PCOS patients than in lean patients, although the difference lacked statistical significance. Comparable results were observed by Layegh et al. (2016) and Siddiqui et al. (2010), whereas Makhija et al. (2023) further pointed out that Polycystic Ovary Syndrome mainly influences women of reproductive age [19-21].

A larger percentage of obese individuals were married and homemakers, while lean individuals exhibited comparatively higher education levels and a larger share of students. Nonetheless, these differences lacked statistical significance, aligning with findings by Kamrul-Hasan and Aalpona (2021), in which sociodemographic factors exhibited no significant variation between obese and lean PCOS populations [22]. The majority of individuals in both groups were part of the middle socioeconomic class, echoing trends noted in local studies like Kamrul-Hasan and Aalpona (2021) [22].

In this study, serum PSA concentrations were notably elevated in obese PCOS patients relative to lean individuals. The box plot revealed a greater median PSA in obese women, reflecting a persistent trend. These results are backed by Ibrahim et al. (2016) and Mardanian and Heidari (2011), who found increased PSA levels in patients with Polycystic Ovary Syndrome [11,23]. Research conducted by Vural et al. (2007) and Rudnicka et al. (2016) additionally showed a positive relationship between PSA and testosterone levels [17,24]. The elevated PSA in obese individuals may result from increased androgen activity linked to obesity and insulin resistance

Obese patients with PCOS exhibited notably elevated systolic and diastolic blood pressure compared to lean individuals, aligning with observations by Usta et al. (2018) and Siddiqui et al. (2010), indicating heightened cardiovascular risk in Polycystic Ovary Syndrome associated with obesity [20,25]. Adjusted Ferriman–Gallwey scores were notably elevated in obese patients, aligning with Layegh et al. (2016), reflecting more severe hyperandrogenism [19]. Obese patients exhibited a significantly higher prevalence of acne vulgaris and acanthosis nigricans, as noted by Li et al. (2012) and Makhija et al. (2023), indicating underlying conditions of insulin resistance and excess androgen [21,26].

Obese PCOS patients exhibited markedly elevated HbA1C and triglyceride levels compared to lean patients, reflecting increased insulin resistance and metabolic disruption. Comparable results were noted by Makhija et al. (2023) and Hussein et al. (2023) concerning Polycystic Ovary Syndrome [21,27]. No notable differences were observed in total cholesterol, HDL, and LDL, aligning with several earlier studies [27].

In this study, serum testosterone levels were notably elevated in obese PCOS patients relative to lean ones, suggesting increased biochemical hyperandrogenism. Comparable results were noted by Usta et al. (2018) and Makhija et al. (2023), reinforcing the link between obesity and elevated androgen levels in Polycystic Ovary Syndrome [21,25].

Nevertheless, no considerable differences were seen in progesterone, TSH, LH, FSH, LH:FSH ratio, and prolactin levels between the two groups. This aligns with results from Li et al. (2012) and Rudnicka et al. (2016), indicating that there were no significant differences in most hormonal parameters between obese and lean PCOS patients [24,26].

Obese PCOS patients exhibited an increased risk of hyperandrogenic traits, with acne vulgaris and acanthosis nigricans occurring significantly more frequently than in lean patients. These findings

coincide with Li et al. (2012) and Makhija et al. (2023), suggesting that obesity enhances insulin resistance and skin symptoms in Polycystic Ovary Syndrome [21,26].

In general, obesity in PCOS individuals is linked to increased hyperandrogenism, metabolic issues, and greater clinical severity than in lean individuals.

## CONCLUSION:

This study demonstrates that obese women with polycystic ovary syndrome (PCOS) exhibit distinct clinical, metabolic, and hormonal profiles compared to their lean counterparts. Obese PCOS patients had significantly higher serum PSA and testosterone levels, elevated blood pressure, greater hirsutism (Modified Ferriman–Gallwey score), and increased prevalence of acne vulgaris and acanthosis nigricans. Metabolic disturbances, including higher HbA1C and triglyceride levels, were also more pronounced in the obese group. In contrast, other hormonal parameters such as progesterone, TSH, LH, FSH, LH:FSH ratio, and prolactin did not differ significantly between the groups. Obesity was strongly associated with dermatological manifestations linked to hyperandrogenism and insulin resistance, particularly acanthosis nigricans.

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