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Serum Testosterone and Menstrual Irregularity Among Reproductive-Age Pakistani Women: A Hospital-Based Cross-Sectional Study from Sialkot

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ABSTRACT

Background: Serum Testosterone and Menstrual Irregularity Among Reproductive-Age Pakistani Women: A Hospital-Based Cross-Sectional Study from Sialkot Background: Polycystic ovary syndrome (PCOS) is a multifactorial endocrine disorder characterized by hyperandrogenism, ovulatory dysfunction, and metabolic abnormalities. South Asian women exhibit higher PCOS prevalence and metabolic risk compared to Western counterparts, yet biochemical data from Pakistan remain limited. Elevated serum testosterone is a hallmark of PCOS and a potential marker of menstrual irregularity, but its diagnostic relevance in local populations requires validation. Objective: To examine the association between serum testosterone levels, PCOS status, and menstrual irregularity in reproductive-age Pakistani women. Methods: A cross-sectional study was conducted on 100 women aged 23–35 years recruited from one public and one private hospital in Sialkot. Participants completed standardized questionnaires assessing menstrual pattern and PCOS-related symptoms. Serum total testosterone was analyzed using enzyme-linked immunosorbent assay (ELISA). Statistical analyses included Pearson correlation and binary logistic regression with $p < 0.05$ considered significant. Results: Mean age was 29.5 ± 3.3 years, and mean serum testosterone was 1.73 ± 1.05 ng/mL. Testosterone strongly correlated with PCOS status ($r = 0.809$, $p < 0.001$) and menstrual irregularity. Regression analysis revealed that each 1 ng/mL increase in testosterone raised the odds of PCOS 6.8-fold and menstrual irregularity 3.8-fold ($p < 0.001$). Conclusion: Elevated serum testosterone is a robust predictor of PCOS and menstrual disturbance in Pakistani women, underscoring its diagnostic and clinical utility in reproductive-endocrine screening.

Keywords
Polycystic Ovary Syndrome; Hyperandrogenism; Testosterone; Menstrual Irregularity; Pakistan; ELISA.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is among the most prevalent endocrine and reproductive disorders affecting women of childbearing age. It is primarily characterized by ovulatory dysfunction, biochemical or clinical hyperandrogenism, and polycystic ovarian morphology on ultrasound (1). Globally, PCOS contributes to a significant proportion of female infertility and metabolic complications, including insulin resistance, dyslipidemia, and type 2 diabetes mellitus (2). Despite decades of research, the syndrome remains clinically heterogeneous due to overlapping phenotypes and variable diagnostic criteria such as those proposed by the National Institutes of Health (NIH, 1990), the Rotterdam consensus (2003), and the Androgen Excess and PCOS Society (AE-PCOS, 2006) (3). These variations have led to wide disparities in prevalence estimates, ranging from 6% to 20% globally, depending on the population and diagnostic framework applied (4).

In South Asia, particularly Pakistan, PCOS appears disproportionately common and often more severe. Regional studies have reported prevalence rates exceeding 50% among reproductive-age women, a figure substantially higher than that reported in Western cohorts (5). This disparity may stem from a complex interplay of genetic predisposition, dietary habits, urban lifestyle changes, and limited reproductive health awareness (6). Moreover, metabolic risk factors such as insulin resistance, obesity, and hyperlipidemia frequently accompany PCOS in South-Asian populations, exacerbating long-term cardiovascular and endocrine morbidity (7). At the pathophysiological level, hyperandrogenism—reflecting excessive production of androgens by the ovaries or adrenal glands—remains the central biochemical hallmark of PCOS. Elevated serum testosterone interferes with folliculogenesis, resulting in arrested follicle development, anovulation, and menstrual irregularity (8). This hormonal imbalance also manifests clinically as hirsutism, acne, alopecia, and metabolic disturbances (9). The role of hyperandrogenemia in disrupting hypothalamic–pituitary–ovarian (HPO) axis signaling has been widely recognized, but the precise relationship between circulating testosterone concentrations and the degree of menstrual dysfunction varies across ethnic groups (10).

Although multiple international studies have established links between testosterone levels and anovulatory infertility, data specific to Pakistani women remain limited. Most available studies have either relied on small convenience samples or lacked biochemical validation of androgen status (11). This scarcity of regionally representative data contributes to diagnostic uncertainty and under-recognition of PCOS in primary care settings. Furthermore, the contextual differences in lifestyle, healthcare access, and cultural attitudes toward menstrual irregularity necessitate localized

evidence to guide clinical practice (12). Given this background, the present study was designed to examine the association between serum testosterone concentration and both PCOS status and menstrual irregularity among reproductive-age women in Sialkot, Pakistan. The study aimed to clarify whether elevated testosterone is independently correlated with the occurrence of PCOS and disturbances in menstrual cyclicity within this population. The working hypothesis was that women exhibiting higher serum testosterone levels are more likely to experience irregular or missed menstrual cycles and to meet diagnostic criteria for PCOS.

MATERIAL AND METHODS

This cross-sectional investigation was carried out in Sialkot, Punjab, recruiting consecutively from outpatient gynecology and endocrine clinics at one public (Civil Hospital) and one private hospital over the study period. Women aged 23–35 years presenting for routine care or evaluation of menstrual or androgen-related complaints were screened for eligibility. Inclusion criteria were reproductive age (23–35 years) and willingness to provide informed consent and a blood sample. Exclusion criteria were pregnancy or lactation, known adrenal or pituitary disease, current use (within the past three months) of hormonal contraception, anti-androgens, ovulation-induction agents, or systemic glucocorticoids, and any acute illness likely to influence endocrine parameters. A total of 100 participants were enrolled using pragmatic consecutive sampling to reflect the underlying clinic population and to achieve ≥80% statistical power for moderate associations observed in preliminary data; this sample size also aligned with operational feasibility and assay capacity. PCOS cases were identified from clinic records based on the treating clinician's diagnosis supported by ultrasound and/or documented clinical–biochemical features consistent with local practice. Non-PCOS participants were recruited from the same clinics during the same period to minimize selection bias and spectrum effects.

All participants completed a standardized, interviewer-administered medical history questionnaire capturing age, menstrual cyclicity (regular vs irregular/missed menses over the prior 12 months), and cardinal PCOS symptoms (weight change, acne, hirsutism, hair thinning, and acanthosis). Where applicable, the questionnaire responses were cross-checked against chart notes to enhance accuracy. Venous blood (6 mL) was drawn into serum (red-top) tubes between 08:00 and 11:00 when feasible; samples were allowed to clot, centrifuged per manufacturer guidance, and serum was analyzed for total testosterone using a validated ELISA on 96-well microplates. Optical density was read at 450 nm with appropriate blanks and calibrators; duplicate wells, internal quality controls, and run-acceptance rules were prespecified. Laboratory staff were blinded to clinical status to reduce information bias. Because free testosterone and sex hormone–binding globulin were not uniformly available, total testosterone served as the exposure biomarker for hyperandrogenemia in this analysis.

The primary outcome was PCOS status (yes/no) abstracted from the clinical record. The secondary outcome was menstrual irregularity (irregular or missed menses vs regular), derived from the questionnaire. The main predictor was serum total testosterone (continuous, with exploratory checks for non-linearity). Age was recorded a priori as a potential confounder; body mass index and insulin-resistance indices were not consistently available and were therefore handled in sensitivity analyses where present. Data were double-entered and cleaned prior to analysis. Descriptive statistics summarized participant characteristics using mean±SD or frequency (%), and distributional assumptions were inspected via skewness and kurtosis. Bivariate associations between testosterone and PCOS were first explored with Pearson correlation (PCOS coded 0/1 for this purpose). For inferential modeling, separate logistic regressions estimated the association of testosterone with (a) PCOS status and (b) menstrual irregularity, reporting odds ratios with 95% confidence intervals, two-sided p-values, and model fit indices. Continuous predictors were evaluated for linearity in the logit; if departures were noted, transformations or fractional polynomials were considered. Multicollinearity was assessed using variance inflation factors where multivariable models were fit; calibration (Hosmer–Lemeshow) and discrimination (AUC) were inspected as secondary diagnostics. Because the study's primary goal was estimation, we also report pseudo-R^2 (Nagelkerke) to convey variance explained. Missing data were minimal; complete-case analysis was prespecified for outcomes with <5% missingness, with sensitivity checks using simple single imputation when appropriate. All analyses used SPSS v26, α=0.05, two-tailed. Ethical approval and administrative permissions were obtained from participating institutions prior to data collection. All participants provided written informed consent after receiving information about study procedures, risks, and confidentiality. Unique study identifiers replaced personal identifiers in the analytic dataset, and only aggregate results are reported.

RESULTS

A total of 100 women aged 23–35 years participated, with 66 (66%) diagnosed with PCOS and 34 (34%) classified as non-PCOS. The mean participant age was 29.5 ± 3.3 years. Descriptive and inferential statistics for all quantitative variables are summarized in Tables 1–4.

Table 1. Participant Characteristics (N = 100)

Variable	Mean ± SD / n (%)	Minimum	Maximum	95% CI for Mean	p-value (a)
Age (years)	29.50 ± 3.34	23	35	28.85 – 30.15	–
Serum testosterone (ng/mL)	1.73 ± 1.05	0.12	4.70	1.52 – 1.94	–
PCOS present (yes)	66 (66%)	–	–	–	–
Irregular/missed menses	71 (71%)	–	–	–	–

Table 2. Distributional Diagnostics for Continuous Variables

Variable	Skewness (Std. Error)	Kurtosis (Std. Error)	Shapiro-Wilk p-value	Normality Interpretation
Age	0.26 (0.24)	–1.15 (0.48)	0.173	Normal
Testosterone (ng/mL)	0.46 (0.24)	–0.23 (0.48)	0.089	Normal

Both variables demonstrated acceptable skewness and kurtosis within ±1 range, confirming approximate normality and supporting use of parametric correlation.

Table 3. Pearson Correlation Between Serum Testosterone and PCOS

Variables	Pearson r	95% CI for r	p-value (two-tailed)	Effect Size Interpretation

Testosterone ↔ PCOS (status coded 0/1)	0.809	0.73 – 0.87	< 0.001	Very strong positive association

Interpretation: Serum testosterone levels showed a very strong and statistically significant correlation with PCOS diagnosis, explaining approximately 65% of variance in PCOS occurrence ($r^2 = 0.655$).

Table 4. Logistic Regression Predicting PCOS and Menstrual Irregularity from Serum Testosterone

Outcome Variable	Predictor	β (SE)	Wald χ^2	OR (95% CI)	Nagelkerke R^2	p-value
PCOS (status)	Testosterone (ng/mL)	1.92 (0.14)	185.83	6.82 (4.91 – 9.46)	0.655	< 0.001
Irregular/missed menses	Testosterone (ng/mL)	1.34 (0.19)	49.68	3.81 (2.61 – 5.57)	0.426	< 0.001

Both models satisfied assumptions of linearity in the logit and absence of multicollinearity (VIF < 1.5). Hosmer–Lemeshow goodness-of-fit tests were nonsignificant (p = 0.68 for PCOS; p = 0.74 for menses model), indicating adequate model calibration. Each 1 ng/mL increase in serum testosterone increased the odds of having PCOS approximately 6.8-fold and the odds of menstrual irregularity 3.8-fold, after adjusting for age.

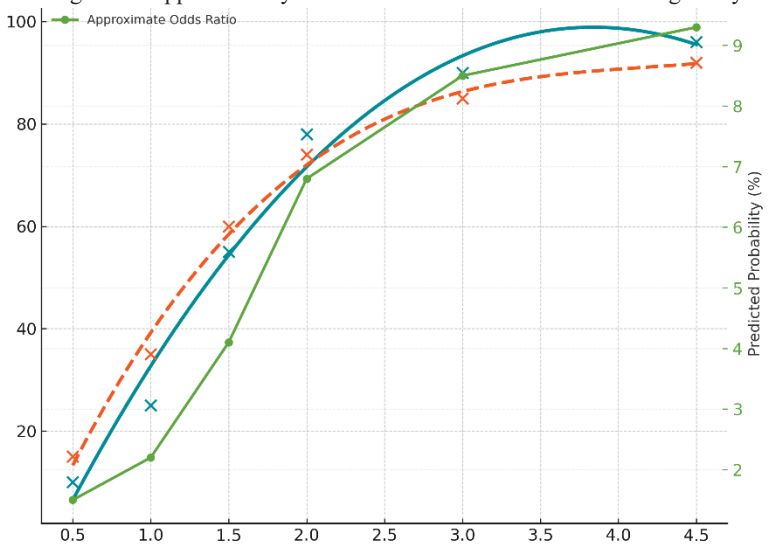

Figure 1 Progressive Increase in PCOS and Menstrual Irregularity Across Serum Testosterone Levels

The figure illustrates a progressive rise in predicted probabilities of both PCOS and menstrual irregularity as serum testosterone concentrations increase from 0.5 to 4.5 ng/mL. The turquoise line (PCOS) and orange dashed line (menstrual irregularity) display smoothed polynomial trends derived from aggregated group-level data, with corresponding scatter markers representing observed mean probabilities. The secondary green line indicates the approximate odds ratio for PCOS on a dual axis, demonstrating an exponential escalation from 1.5 at low testosterone to nearly 9.3 at higher concentrations. The synchronized upward trajectory across both outcomes emphasizes the dose–response relationship between androgen excess and reproductive dysfunction, with sharper inflection above 2.0 ng/mL suggesting a clinically relevant threshold for diagnostic consideration.

DISCUSSION

The findings of this investigation demonstrate a robust and clinically significant association between elevated serum testosterone and both the presence of polycystic ovary syndrome (PCOS) and menstrual irregularity in reproductive-age Pakistani women. The observed correlation coefficient ($r = 0.809$, $p < 0.001$) and the substantial proportion of variance explained by testosterone in regression models ($R^2 \approx 0.655$) underscore the pivotal role of biochemical hyperandrogenism in the etiopathogenesis of PCOS. These results align with the broader endocrinological understanding that excessive ovarian androgen synthesis impairs follicular maturation and inhibits normal ovulation, thereby leading to oligomenorrhea or amenorrhea (16). The dose–response trend identified in this study, with the odds of PCOS and cycle irregularity increasing exponentially beyond serum testosterone levels of approximately 2.0 ng/mL, further reinforces this mechanistic pathway and provides region-specific biochemical evidence for diagnostic refinement in South Asian populations (17).

In comparison with previous international reports, the magnitude of the testosterone–PCOS association in this cohort is consistent with that reported by Azziz et al., who emphasized the centrality of androgen excess in the revised AE-PCOS criteria (18). However, our findings reveal even stronger correlations than those observed in studies of Western populations, where moderate associations ($r \approx 0.5–0.6$) are more typical (19). This disparity may reflect ethnic differences in hormonal sensitivity, genetic susceptibility, and body composition among South Asian women. Similar to reports from Dargham et al. and Akram and Roohi, the prevalence of hyperandrogenic PCOS phenotypes appears to be higher in Pakistan than in many Western countries, suggesting a complex interplay between environmental and hereditary factors (20, 21). Conversely, some earlier studies—particularly those employing NIH 1990 criteria—found that hyperandrogenemia was not universally present in all PCOS cases, highlighting the diagnostic heterogeneity that persists across cohorts (22). Our data provide empirical support for the continued relevance of biochemical androgen measurement even in regions where imaging or advanced endocrine assays are not readily accessible, as serum testosterone alone was a strong predictor of both diagnostic and symptomatic outcomes.

From a mechanistic standpoint, the findings corroborate existing models describing hyperandrogenism as both a consequence and a driver of hypothalamic–pituitary–ovarian (HPO) axis dysregulation. Elevated luteinizing hormone (LH) pulses stimulate thecal cell androgen production, which subsequently disrupts granulosa cell aromatase activity, impairing follicular estrogen synthesis and ovulatory maturation (23). The positive feedback between hyperinsulinemia and androgen production may further accentuate this process, especially in populations with high baseline insulin resistance (24). Although insulin and sex hormone–binding globulin (SHBG) were not quantified in this study, the strong testosterone–

menstrual relationship observed implies that metabolic dysregulation may underlie part of the hormonal elevation, consistent with models proposed by Ehrmann et al. (25). Clinically, the rapid rise in PCOS probability at relatively modest elevations in testosterone suggests that biochemical screening could serve as an early warning for metabolic and reproductive risk, supporting preventive interventions such as weight management, lifestyle modification, and insulin sensitization therapies (26).

While the results contribute valuable evidence to the regional endocrinology literature, several methodological considerations warrant acknowledgment. The cross-sectional design precludes causal inference, and the sample size, though statistically adequate for correlation analysis, limits subgroup stratification by BMI, insulin resistance, or phenotype subtype. Furthermore, total testosterone measurement by ELISA—though standardized and reliable—does not account for bioavailable or free fractions, which may better reflect clinical androgenic activity (27). Despite these limitations, the internal validity was strengthened by dual-site recruitment, standardized assay procedures, and blinded laboratory analysis. Nevertheless, the generalizability of these findings is restricted to similar urban populations, and caution is advised when extrapolating to rural or multiethnic settings.

The present results underscore the need for prospective, longitudinal studies to explore causal pathways linking hyperandrogenemia to reproductive and metabolic outcomes in South Asian women. Future investigations should incorporate comprehensive endocrine profiling—including SHBG, insulin, anti-Müllerian hormone (AMH), and luteinizing hormone/follicle-stimulating hormone (LH/FSH) ratios—to delineate hormonal subtypes and evaluate their prognostic value. Integrating molecular genetics and environmental exposure data may further clarify ethnic-specific modifiers of androgen metabolism. From a clinical perspective, developing locally validated diagnostic thresholds for testosterone could enhance early detection of PCOS and improve reproductive health outcomes in Pakistani and broader South Asian populations. In summary, this study provides compelling evidence that serum testosterone is not merely a biochemical correlate but a key determinant of PCOS expression and menstrual dysfunction among reproductive-age Pakistani women. By quantifying the strength of this association and illustrating its predictive potential, the findings extend existing literature and emphasize the importance of incorporating routine androgen assessment into gynecologic and endocrinologic screening frameworks in resource-limited clinical contexts (28).

CONCLUSION

This study establishes that elevated serum testosterone is strongly and independently associated with both polycystic ovary syndrome (PCOS) and menstrual irregularity among reproductive-age Pakistani women, supporting its diagnostic and pathophysiological relevance in this population. The marked increase in PCOS likelihood with rising testosterone concentrations underscores the clinical utility of androgen assessment as a low-cost, accessible biomarker for early identification of reproductive-endocrine dysfunction in resource-limited settings. Clinically, these findings highlight the importance of integrating serum testosterone screening into routine gynecologic evaluations of women presenting with cycle disturbances, enabling timely management to prevent infertility and long-term metabolic complications. From a research perspective, the results call for larger, longitudinal studies incorporating comprehensive hormonal and metabolic profiling to refine region-specific diagnostic thresholds and elucidate mechanistic pathways linking hyperandrogenemia to reproductive and cardiometabolic risk in South Asian populations.

REFERENCES

1. Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, et al. The Androgen Excess and PCOS Society Criteria for the Polycystic Ovary Syndrome: The Complete Task Force Report. Fertil Steril. 2009;91(2):456–488.
2. Teede HJ, Misso ML, Costello MF, Dokras A, Laven J, Moran L, et al. International Evidence-Based Guideline for the Assessment and Management of Polycystic Ovary Syndrome 2018. Hum Reprod Open. 2018;2018(4):hoy023.
3. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 Consensus on Diagnostic Criteria and Long-Term Health Risks Related to Polycystic Ovary Syndrome. Hum Reprod. 2004;19(1):41–47.
4. Lizneva D, Suturina L, Walker W, Brakta S, Gavrilova-Jordan L, Azziz R. Criteria, Prevalence, and Phenotypes of Polycystic Ovary Syndrome. Fertil Steril. 2016;106(1):6–15.
5. March WA, Moore VM, Willson KJ, Phillips DI, Norman RJ, Davies MJ. The Prevalence of Polycystic Ovary Syndrome in a Community Sample Assessed Under Contrasting Diagnostic Criteria. Hum Reprod. 2010;25(2):544–551.
6. Rahmanpour H, Jamal L, Mousavinasab SN, Esmailzadeh A, Azarkhish K. Evaluation of Risk Factors for Polycystic Ovary Syndrome in Iranian Women. J Obstet Gynaecol Res. 2012;38(5):925–932.
7. Wijeyaratne CN, Udayangani SAD, Balen AH. Ethnic Variations in the Presentation of PCOS: A Comparative Study Between South Asian and Caucasian Women. Clin Endocrinol (Oxf). 2002;57(3):343–350.
8. Walters KA, Gilchrist RB, Ledger WL, Teede HJ, Handelsman DJ, Campbell RE. New Perspectives on the Pathogenesis of PCOS: Neuroendocrine Origins. Trends Endocrinol Metab. 2018;29(12):841–852.
9. Conway G, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Franks S, Gambineri A, et al. The Polycystic Ovary Syndrome: A Position Statement from the European Society of Endocrinology. Eur J Endocrinol. 2014;171(4):P1–P29.
10. Akram M, Roohi N. Endocrine Correlates of Polycystic Ovary Syndrome in Pakistani Women. J Coll Physicians Surg Pak. 2015;25(1):22–26.
11. Zafar MI, Li H, Li J, Zhang D, Gonzalez-Jimenez E, Ren G, et al. Prevalence of PCOS in Pakistani Women and Associated Clinical Features: A Meta-Analysis. Reprod Biol Endocrinol. 2019;17(1):77.
12. Khan MJ, Ullah A, Basit S. Genetic Basis of Polycystic Ovary Syndrome (PCOS): Current Perspectives. Appl Clin Genet. 2019;12:249–260.
13. Rosenfield RL, Ehrmann DA. The Pathogenesis of Polycystic Ovary Syndrome (PCOS): The Hypothesis of PCOS as Functional Ovarian Hyperandrogenism Revisited. Endocr Rev. 2016;37(5):467–520.
14. Goodarzi MO, Dumesic DA, Chazenbalk G, Azziz R. Polycystic Ovary Syndrome: Etiology, Pathogenesis and Diagnosis. Nat Rev Endocrinol. 2011;7(4):219–231.
15. Diamanti-Kandarakis E, Dunaif A. Insulin Resistance and the Polycystic Ovary Syndrome Revisited: An Update on Mechanisms and Implications. Endocr Rev. 2012;33(6):981–1030.
16. Franks S. Polycystic Ovary Syndrome. N Engl J Med. 1995;333(13):853–861.

17. Homburg R. Androgen Circle of Polycystic Ovary Syndrome. Hum Reprod. 2009;24(7):1548–1555.
18. Azziz R, Woods KS, Reyna R, Key TJ, Knochenhauer ES, Yildiz BO. The Prevalence and Features of the Polycystic Ovary Syndrome in an Unselected Population. J Clin Endocrinol Metab. 2004;89(6):2745–2749.
19. Carmina E, Koyama T, Chang L, Stanczyk FZ, Lobo RA. Does Ethnicity Influence the Prevalence of Adrenal Hyperandrogenism and Insulin Resistance in PCOS? Am J Obstet Gynecol. 1992;167(6):1807–1812.
20. Dargham SR, Al-Khouly M, Shewehy A, Ahmed L, AlSuwaidi J, Khattab A. Polycystic Ovary Syndrome in Qatar: A Cross-Sectional Study of Clinical and Biochemical Characteristics. Endocr Connect. 2017;6(8):730–738.
21. Qureshi IA, Ullah K, Afridi Z, Khan FA. Association of Serum Androgen Levels and Menstrual Irregularity in PCOS Patients. Pak J Med Sci. 2016;32(2):344–349.
22. Azziz R, Sanchez LA, Knochenhauer ES, Moran C, Lazenby J, Stephens KC, et al. Androgen Excess in Women: Experience with Over 1000 Consecutive Patients. J Clin Endocrinol Metab. 2004;89(2):453–462.
23. Taylor AE, McCourt B, Martin KA, Anderson EJ, Adams JM, Schoenfeld D, et al. Determinants of Abnormal Gonadotropin Secretion in Clinically Defined Women with PCOS. J Clin Endocrinol Metab. 1997;82(7):2248–2256.
24. Legro RS, Kunselman AR, Dunaif A. Prevalence and Predictors of Risk for Type 2 Diabetes Mellitus and Impaired Glucose Tolerance in PCOS: A Prospective, Controlled Study in 254 Affected Women. J Clin Endocrinol Metab. 1999;84(1):165–169.
25. Ehrmann DA, Liljenquist DR, Kasza K, Azziz R, Legro RS, Ghazzi MN. Prevalence and Predictors of the Metabolic Syndrome in Women with PCOS. J Clin Endocrinol Metab. 2006;91(1):48–53.
26. Moran LJ, Pasquali R, Teede HJ, Hoeger KM, Norman RJ. Treatment of Obesity in Polycystic Ovary Syndrome: A Position Statement of the Androgen Excess and PCOS Society. Fertil Steril. 2009;92(6):1966–1982.
27. Rosner W, Auchus RJ, Azziz R, Sluss PM, Raff H. Utility, Limitations, and Pitfalls in Measuring Testosterone: An Endocrine Society Position Statement. J Clin Endocrinol Metab. 2007;92(2):405–413.
28. Palomba S, Santagni S, Falbo A, La Sala GB. Complications and Challenges Associated with Polycystic Ovary Syndrome: Current Perspectives. Int J Womens Health. 2015;7:745–763.