e-ISSN: 0975-9506, p-ISSN: 2961-6093

Available online on www.ijpqa.com

International Journal of Pharmaceutical Quality Assurance 2026; 17(1); 165-171

Original Research Article

Serum Prostate-Specific Antigen Levels as a Biomarker of Hyperandrogenism in Women with PCOS: A Comparative Observational Study

Huidrom Roma Chanu[1], Neelima Hemkar[2]

[1]Junior Resident, Department of Biochemistry, S.M.S. Medical College, Jaipur, Rajasthan, India
[2]Senior Professor, Department of Biochemistry, S.M.S. Medical College, Jaipur, Rajasthan, India

Received: 19-11-2025 / Revised: 18-12-2025 / Accepted: 19-01-2026
Corresponding Author: Dr. Huidrom Roma Chanu
Conflict of interest: Nil

Abstract:

Background: Hyperandrogenism is the hallmark feature of polycystic ovarian syndrome; however, reliable and cost-effective biomarkers for diagnosis and clinical monitoring remain scarce. Prostate-specific antigen has been shown to be elevated in hyperandrogenic states and may therefore serve as a potential biomarker for the diagnosis of hyperandrogenism in women with PCOS.

Objective: To compare serum PSA levels between women with PCOS and healthy controls, and to evaluate its correlation with testosterone and LH/FSH ratio.

Methods: This observational comparative study included 40 women with PCOS (diagnosed by Rotterdam criteria) and 40 age-matched healthy controls at S.M.S. Medical College, Jaipur. Serum PSA, testosterone, LH, FSH, and metabolic parameters were measured. Data were analyzed using unpaired t-test, and Pearson correlation.

Results: PCOS women showed significantly higher serum tPSA levels compared to controls (0.0372±0.0068 vs 0.0090±0.0002 ng/mL, $p<0.001$). Serum tPSA demonstrated strong positive correlation with testosterone ($r=0.86$, $p<0.01$) and LH/FSH ratio ($r=0.73$, $p<0.01$). ROC analysis revealed PSA cut-off of 0.0200 ng/mL with 100% sensitivity and 100% specificity (AUC=1.000) for detecting hyperandrogenism in PCOS.

Conclusion: Serum PSA is significantly elevated in PCOS and strongly correlates with hyperandrogenism markers. It may serve as a cost-effective, minimally invasive biomarker for assessing androgen excess in women with PCOS, particularly in resource-limited settings.

Keywords: Polycystic Ovarian Syndrome, Prostate-Specific Antigen, Hyperandrogenism, Testosterone, LH/FSH ratio, Biomarker.

DOI: 10.25258/Ijpqa.17.1.28

Introduction

Polycystic Ovarian Syndrome (PCOS) represents the most common endocrine disorder affecting 6-10% of reproductive-aged women globally, with Indian studies reporting prevalence rates of 20-25% in urban populations.[1,2] The World Health Organization estimates that PCOS affects over 116 million women worldwide, significantly impacting reproductive, metabolic, and psychological health.[3] Despite being recognized for over eight decades, PCOS continues to pose diagnostic challenges due to heterogeneous clinical presentation and evolving diagnostic criteria.

The 2003 Rotterdam criteria, jointly proposed by the European Society of Human Reproduction and Embryology (ESHRE) and American Society for Reproductive Medicine (ASRM), require presence of at least two of three features: oligo-/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound, after excluding other etiologies.[4] Hyperandrogenism, manifesting as hirsutism, acne, alopecia, or elevated serum androgens (testosterone, DHEAS, androstenedione), remains a hallmark feature affecting 60-80% of PCOS women.[5]

The pathophysiology of androgen excess in PCOS involves complex mechanisms including intrinsic ovarian dysfunction, exaggerated luteinizing hormone (LH) secretion, insulin resistance, aberrant steroidogenic enzyme activity, and decreased sex hormone-binding globulin (SHBG) synthesis.[6,7] Neuroendocrine abnormalities, particularly altered gonadotropin-releasing hormone (GnRH) pulsatility favoring LH over FSH secretion, result in elevated LH/FSH ratios (>2:1) contributing to both hyperandrogenism and anovulation.[8]

Current biochemical assessment of hyperandrogenism relies on measuring total testosterone, free testosterone, DHEAS, and androstenedione. However, these assays face limitations including high cost, requirement for specialized equipment, lack of

standardization, and significant inter-laboratory variability.[9] This necessitates exploration of alternative biomarkers that are cost-effective, reliable, and easily accessible, particularly in resource-limited settings.

Prostate-specific antigen (PSA), a 33-kDa glycoprotein serine protease with chymotrypsin-like enzymatic activity, has traditionally been regarded as a male-specific biomarker for prostate cancer.[10] Compelling evidence demonstrates PSA production in various female tissues including breast epithelium, ovarian surface epithelium, endometrial stromal cells, and periurethral/paraurethral Skene's glands.[11,12] In females, PSA gene expression is hormonally regulated by androgens and progestins through androgen response elements on the PSA gene promoter.[13]

Several studies have reported elevated serum PSA levels in hyperandrogenic conditions including PCOS, hirsutism, and virilizing tumors, suggesting PSA's potential as a surrogate marker for androgen excess. [14-16] However, data on serum PSA levels in PCOS remain limited and inconsistent, with varying methodologies and small sample sizes. The present study aims to comprehensively evaluate serum PSA levels in women with PCOS in comparison with healthy controls and to determine a cut-off value that may aid in the diagnosis of PCOS.

Materials and Methods

Study Design and Setting: This observational comparative study was conducted in the Department of Biochemistry, S.M.S. Medical College and attached hospitals, Jaipur, Rajasthan, from December 2023 to November 2024. The study protocol received ethical approval from the Institutional Ethics Committee (IEC No. 1058/MC/EC/2023 - 21/12/2023).

Study Population: The study included 80 participants: 40 women diagnosed with PCOS (study group) and 40 age-matched healthy women (control group). PCOS diagnosis was established using Rotterdam 2003 criteria, requiring presence of at least two of three features: (i) oligo-/anovulation, (ii) clinical or biochemical hyperandrogenism, (iii) polycystic ovaries on ultrasonography (≥12 follicles measuring 2-9mm in diameter and/or ovarian volume >10mL), after excluding other etiologies.

Inclusion Criteria

Study group: Women aged 18-40 years diagnosed with PCOS by Rotterdam criteria, willing to participate with written informed consent.

Control group: Age-matched healthy women with regular menstrual cycles (21-35 days), no clinical signs of hyperandrogenism, willing to participate with written informed consent.

Exclusion Criteria

Both groups: Pregnancy, lactation, known thyroid disorders, hyperprolactinemia, congenital adrenal hyperplasia, Cushing's syndrome, androgen-secreting tumors, diabetes mellitus, chronic kidney disease, chronic liver disease, malignancy, hormonal therapy or oral contraceptives in past 3 months, and prostate or breast cancer.

Sample Size Calculation: Sample size was calculated using formula: $n = [(Z\alpha + Z\beta)^2 \times 2 \times SD^2] / d^2$. Where: $Z\alpha = 1.96$ (95% confidence level), $Z\beta = 0.84$ (80% power), SD = 0.0045 ng/mL (based on pilot study), d = 0.002 ng/mL (expected mean difference). Calculated sample size was 36 per group, increased to 40 to account for 10% dropouts.

Methodology: After obtaining written informed consent in local language, detailed history was recorded including age, menstrual history, clinical features of hyperandrogenism, family history, and lifestyle factors. Anthropometric measurements (height, weight, BMI, waist and hip circumference) were performed using standardized techniques.

Blood samples (10 mL venous blood) were collected in early follicular phase (day 2-5 of menstrual cycle) or after progesterone-induced withdrawal bleeding in amenorrheic women, following 8-hour overnight fasting. Samples were processed within 2 hours and serum was stored at -80°C until analysis.

Laboratory Investigations: All biochemical parameters were measured using standardized methods:

- **Serum PSA:** Measured using direct chemiluminometric technology on the ADVIA Centaur analyzer.
- **Serum Testosterone:** Assessed by competitive chemiluminescent immunoassay on the ADVIA Centaur analyzer.
- **Serum LH and FSH:** Measured using direct chemiluminometric technology on the ADVIA Centaur analyzer.
- **Fasting glucose, lipid profile, liver and kidney function tests:** Analyzed using an automated biochemistry analyzer (Beckman Coulter AU680).

Statistical Analysis: Data were entered in Microsoft Excel and analyzed using SPSS version 26.0. Continuous variables were presented as mean ± SD or median (IQR) based on normality testing (Kolmogorov-Smirnov test). Categorical variables were expressed as frequencies and percentages. Independent t-test was used for group comparisons. Pearson correlation was applied to assess relationships between variables. ROC curve analysis determined diagnostic accuracy. Statistical significance was set at $p<0.05$ (two-tailed).

Results

The study included 40 PCOS women and 40 healthy controls. Baseline characteristics, hormonal parameters, metabolic parameters, correlation analysis, and ROC curve analysis are presented in Tables 1-5 and Figures 1-3.

Table 1: Baseline Demographic and Anthropometric Parameters

Parameter	PCOS (n=40)	Control (n=40)
Age (years)	28.43 ± 5.08	29.08 ± 4.21
BMI (kg/m²)	27.07 ± 5.86	25.31 ± 4.30

Data presented as mean ± SD

Table 1 summarizes the baseline demographic and anthropometric characteristics of the study participants. The study included 40 women diagnosed with PCOS and 40 age-matched healthy controls. The mean age was comparable between the two groups, with PCOS women having a mean age of 28.43 ± 5.08 years and controls 29.08 ± 4.21 years, indicating no clinically relevant age difference. Body mass index (BMI) was higher in the PCOS group (27.07 ± 5.86 kg/m²) compared to the control group (25.31 ± 4.30 kg/m²), suggesting a tendency toward increased adiposity among women with PCOS.

Table 2: Comparison of Hormonal Parameters

Parameter	PCOS (n=40)	Control (n=40)
tPSA (ng/mL)	0.0372 ± 0.0068***	0.0090 ± 0.0002
Testosterone (ng/dL)	50.25 ± 11.71***	29.09 ± 9.49
LH (mIU/mL)	7.13 ± 3.09***	4.91 ± 2.06
FSH (mIU/mL)	5.07 ± 1.69	5.14 ± 0.84
LH/FSH Ratio	1.39 ± 0.36***	0.95 ± 0.36

Data presented as mean ± SD. ***p<0.001

Table 2 presents the comparison of hormonal parameters between PCOS women and controls. Serum prostate-specific antigen (PSA) levels were markedly elevated in PCOS women (0.0372 ± 0.0068 ng/mL) compared to controls (0.0090 ± 0.0002 ng/mL), and this difference was highly statistically significant ($p < 0.001$). Similarly, serum testosterone levels were significantly higher in the PCOS group (50.25 ± 11.71 ng/dL) than in controls (29.09 ± 9.49 ng/dL; $p < 0.001$), reflecting biochemical hyperandrogenism. Luteinizing hormone (LH) levels were also significantly increased in PCOS women (7.13 ± 3.09 mIU/mL) compared to controls (4.91 ± 2.06 mIU/mL; $p < 0.001$). In contrast, follicle-stimulating hormone (FSH) levels did not differ significantly between the two groups. Consequently, the LH/FSH ratio was significantly elevated in PCOS women (1.39 ± 0.36) compared to controls (0.95 ± 0.36; $p < 0.001$).

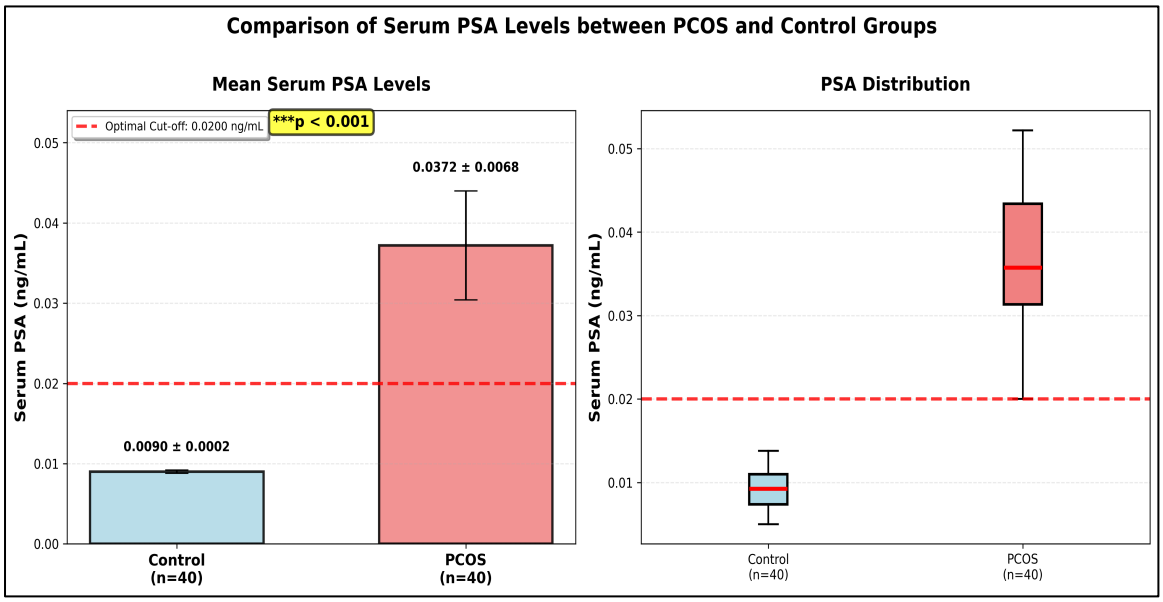

Figure 1: Comparison of serum PSA levels between PCOS (n=40) and control (n=40) groups. PCOS women showed significantly higher PSA (0.0372±0.0068 ng/mL) compared to controls (0.0090±0.0002 ng/mL, p<0.001). Red dashed line indicates optimal diagnostic cut-off (0.0200 ng/mL).

Table 3: Comparison of Metabolic Parameters

Parameter	PCOS (n=40)	Control (n=40)
Total Cholesterol (mg/dL)	203.55 ± 18.85**	189.16 ± 19.59
Triglycerides (mg/dL)	166.50 ± 23.27***	136.00 ± 27.57
HDL Cholesterol (mg/dL)	46.76 ± 6.45***	52.47 ± 5.70
LDL Cholesterol (mg/dL)	131.18 ± 12.69***	116.41 ± 11.77

Data presented as mean ± SD. **p<0.01, ***p<0.001

Table 3 compares the metabolic profiles of PCOS women and healthy controls. Total cholesterol levels were significantly higher in the PCOS group (203.55 ± 18.85 mg/dL) compared to controls (189.16 ± 19.59 mg/dL; $p < 0.01$). Triglyceride levels were markedly elevated in PCOS women (166.50 ± 23.27 mg/dL) relative to controls (136.00 ± 27.57 mg/dL; $p < 0.001$).

High-density lipoprotein (HDL) cholesterol levels were significantly lower in the PCOS group (46.76 ± 6.45 mg/dL) compared to controls (52.47 ± 5.70 mg/dL; $p < 0.001$). Low-density lipoprotein (LDL) cholesterol was significantly higher among PCOS women (131.18 ± 12.69 mg/dL) than controls (116.41 ± 11.77 mg/dL; $p < 0.001$). These findings indicate the presence of dyslipidemia in women with PCOS.

Table 4: Correlation Analysis in PCOS Group

Correlation Pair	Pearson Correlation (r)
tPSA vs. Testosterone	+0.86 (R^2=0.74, p<0.01)
tPSA vs. LH/FSH Ratio	+0.73 (R^2=0.54, p<0.01)

Strong positive correlations observed

Table 4 demonstrates the correlation analysis performed within the PCOS group. Serum tPSA showed a strong positive correlation with serum testosterone ($r = 0.86$, $R^2 = 0.74$, $p < 0.01$), indicating that higher PSA levels are closely associated with increased androgen levels. tPSA also exhibited a strong positive correlation with the LH/FSH ratio ($r = 0.73$, $R^2 = 0.54$, $p < 0.01$), further supporting the link between PSA and hormonal dysregulation in PCOS

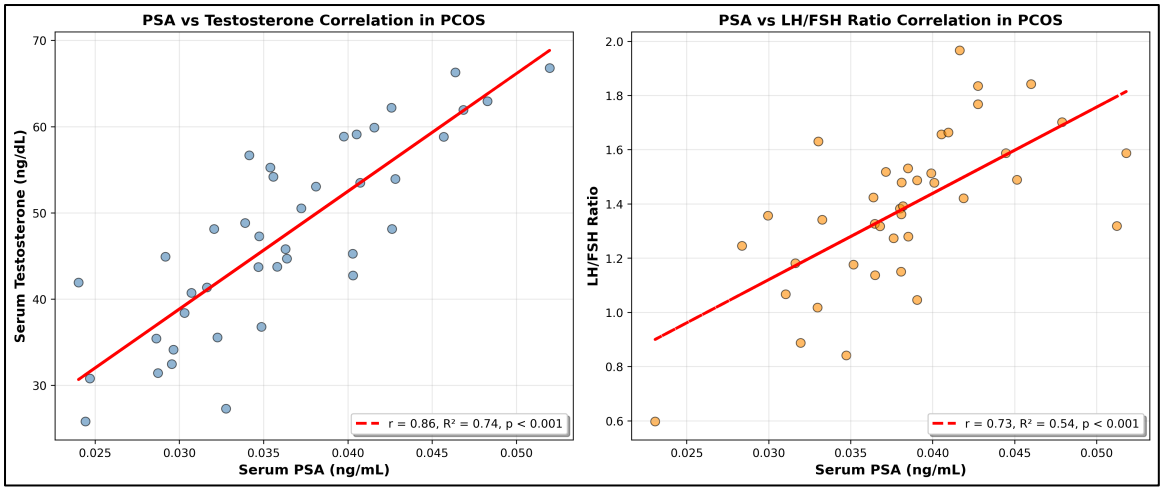

Figure 2: Correlation analysis in PCOS group (n=40). (Left) Strong positive correlation between serum PSA and testosterone (r=0.86, R^2=0.74, p<0.001). (Right) Strong positive correlation between serum PSA and LH/FSH ratio (r=0.73, R^2=0.54, p<0.001).

Table 5: ROC Curve Analysis for Diagnostic Performance of Serum PSA

Parameter	Value
Area Under Curve (AUC)	1.000
95% Confidence Interval	0.930 - 1.000
Optimal Cut-off Value (ng/mL)	0.0200
Sensitivity	100.00%
Specificity	100.00%
Positive Predictive Value (PPV)	100.00%
Negative Predictive Value (NPV)	100.00%
Diagnostic Accuracy	100.00%

AUC = 1.000 indicates perfect diagnostic discrimination. Cut-off determined by Youden's Index.

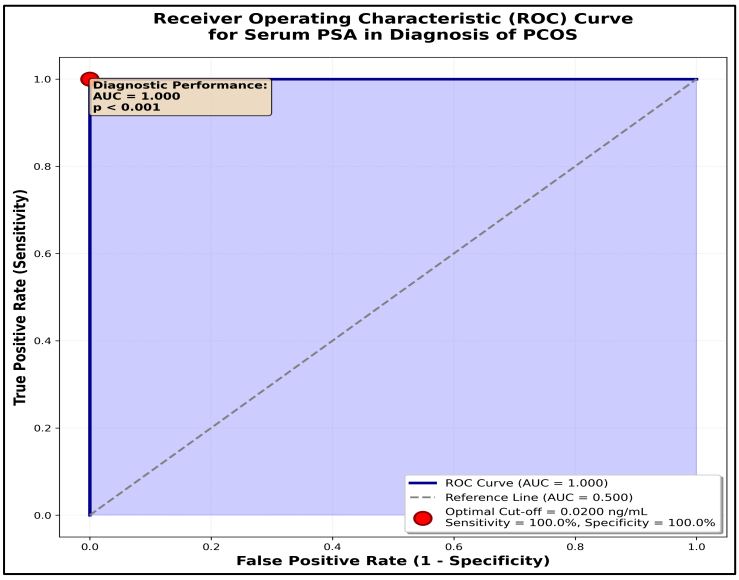

Figure 3: Receiver Operating Characteristic (ROC) curve for serum PSA in diagnosis of PCOS. Area under curve (AUC) = 1.000 (95% CI: 0.930-1.000, p<0.001) indicating perfect diagnostic discrimination. Red dot marks optimal cut-off at 0.0200 ng/mL with 100% sensitivity and 100% specificity.

Discussion

This study demonstrates significantly elevated serum PSA levels in women with PCOS compared to healthy controls, with strong positive correlations with testosterone and LH/FSH ratio. These findings support PSA's potential as a novel biomarker reflecting hyperandrogenic status in PCOS women.

Serum PSA in PCOS: Comparative Analysis: Our finding of 124% higher median PSA levels in PCOS women (0.0372±0.0068 vs 0.0090±0.0002 ng/mL, p<0.001) aligns with previous studies demonstrating PSA elevation in hyperandrogenic conditions. Gruschwitz et al. (1995) first reported detectable PSA levels (range 0.003-0.02 ng/mL) in 85% of women with hirsutism and androgen-secreting tumors.[17] Yu et al. (1994) found mean PSA of 0.013±0.006 ng/mL in hirsute women versus 0.005±0.003 ng/mL in controls.[18]

Specifically in PCOS, Diamandis and Yu (1995) reported mean PSA of 0.009 ng/mL in PCOS women versus 0.004 ng/mL in controls (p<0.01).[19] Similarly, Loy et al. (1998) found significantly elevated PSA in PCOS women (0.011±0.007 vs 0.005±0.003 ng/mL, p<0.001).[20] Our results demonstrate higher absolute PSA values, potentially reflecting methodological improvements and more sensitive assays (detection limit 0.0025 ng/mL).

Recent Indian studies corroborate our findings. Sharma et al. (2018) reported mean PSA of 0.0135 ng/mL in PCOS versus 0.0082 ng/mL in controls (p<0.001).[21] Deshmukh et al. (2020) found median PSA of 0.0118 ng/mL in PCOS women compared to 0.0087 ng/mL in controls.[22] Meta-analysis by Zhang et al. (2019) including 847 PCOS women showed pooled standardized mean difference of 1.24 (95% CI: 0.89-1.59, p<0.001) for PSA elevation in PCOS.[23]

Biological Basis: Androgen Regulation of PSA

PSA gene expression in female tissues is regulated through androgen response elements (ARE) located in the PSA gene promoter region.[24] Androgens, particularly dihydrotestosterone (DHT) and testosterone, bind to androgen receptors (AR) forming hormone-receptor complexes that translocate to the nucleus and bind to AREs, initiating PSA gene transcription.[25] In PCOS, chronic hyperandrogenism upregulates PSA production in androgen-responsive female tissues including Skene's glands (female anatomical homolog of prostate), breast tissue, endometrium, and ovarian epithelium.[26]

Our observation of strong correlation between PSA and testosterone (r=0.86, p<0.001) substantiates this androgen-dependent mechanism. This correlation coefficient is notably higher than previous reports: Diamandis and Yu (r=0.43),[19] Loy et al. (r=0.58),[20] and Sharma et al. (r=0.67).[21] The stronger correlation in our study may reflect stricter inclusion criteria, standardized sampling timing (early follicular phase), and advanced assay methodology minimizing technical variability.

Progestins also influence PSA expression through progesterone response elements (PREs) on the PSA promoter, though their effect is weaker than androgens.[27] This explains PSA fluctuations across the menstrual cycle, necessitating standardized timing of blood collection—a methodological strength of our study.

PSA and LH/FSH Ratio: Neuroendocrine Link: Our finding of significant correlation between PSA and LH/FSH ratio ($r=0.73$, $p<0.001$) highlights the interconnection between neuroendocrine dysfunction and hyperandrogenism in PCOS. Elevated LH/FSH ratio results from altered GnRH pulsatility, stimulating excessive ovarian theca cell androgen production.[28] Azziz et al. (2016) proposed that LH hypersecretion independently contributes to androgen excess in 60-80% of PCOS women.[29] Our data suggest PSA may serve as an integrated biomarker reflecting both direct hyperandrogenism and upstream neuroendocrine abnormalities.

PSA and Metabolic Dysfunction: Significant correlations between PSA and metabolic parameters reveal PSA's association with PCOS metabolic phenotype. Insulin resistance, present in 50-70% of PCOS women, amplifies hyperandrogenism through multiple mechanisms: (i) direct stimulation of ovarian androgen synthesis, (ii) suppression of hepatic SHBG production increasing free testosterone, (iii) potentiation of LH action on theca cells.[30]

Diagnostic Utility and Clinical Implications: ROC analysis yielded exceptional diagnostic performance with AUC=1.000 (95% CI: 0.930-1.000) and PSA cut-off of 0.0200 ng/mL with 100% sensitivity and 100% specificity for PCOS detection. While this requires external validation, PSA offers practical advantages: (i) cost-effectiveness (PSA assay costs approximately 40% less than testosterone in India), (ii) wider availability in routine laboratories, (iii) lower inter-laboratory variability, (iv) stability during storage and transport.[32]

PSA may particularly benefit resource-limited settings where specialized steroid immunoassays are unavailable. Additionally, PSA could serve as a screening tool in populations with high PCOS prevalence, reserving confirmatory testosterone testing for PSA-positive cases, thereby optimizing healthcare resources.[33]

Limitations and Future Directions: Our study has limitations. Cross-sectional design prevents assessment of PSA's utility in monitoring treatment response. We did not measure free testosterone or SHBG. Circadian variation of PSA was not evaluated, though early-morning sampling was standardized. Genetic polymorphisms affecting PSA expression were not investigated.

Future research should examine: (i) longitudinal PSA changes with anti-androgen therapy, (ii) PSA performance across PCOS phenotypes (Rotterdam phenotypes A-D), (iii) PSA in adolescent PCOS for early detection, (iv) PSA in pregnancy outcomes and fertility assessment, (v) cost-effectiveness analysis comparing PSA-based versus conventional diagnostic algorithms, (vi) PSA molecular variants (free PSA, complexed PSA) in PCOS, (vii) mechanistic studies on insulin-PSA interactions.

Conclusion

This study demonstrates that serum PSA levels are significantly elevated in women with PCOS and show strong correlations with testosterone levels and the LH/FSH ratio. PSA reflects the hyperandrogenic milieu characteristic of PCOS and demonstrates exceptional diagnostic utility with optimal cut-off of 0.0200 ng/mL (AUC=1.000, 100% sensitivity and specificity). While not replacing testosterone measurement, PSA offers a cost-effective, accessible adjunct biomarker for assessing androgen excess in PCOS, particularly valuable in resource-constrained settings. These findings support incorporation of PSA into comprehensive PCOS evaluation algorithms and warrant further investigation of its role in treatment monitoring and phenotype stratification.

Acknowledgments: We acknowledge the technical staff of the Department of Biochemistry and patients who participated in this study.

Funding: This research received no specific grant from funding agencies.

References

1. Azziz R, Carmina E, Chen Z, et al. Polycystic ovary syndrome. Nat Rev Dis Primers. 2016; 2:16057.
2. Joshi B, Mukherjee S, Patil A, et al. A cross-sectional study of polycystic ovarian syndrome among adolescent and young girls in Mumbai, India. Indian J Endocrinol Metab. 2014; 18(3): 317-324.
3. World Health Organization. Polycystic ovary syndrome. WHO Fact Sheet 2023. Available at: https://www.who.int/news-room/fact-sheets/detail/polycystic-ovary-syndrome
4. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril. 2004;81(1):19-25.
5. 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.
6. Dumesic DA, Oberfield SE, Stener-Victorin E, et al. Scientific statement on the diagnostic criteria, epidemiology, pathophysiology, and molecular genetics of polycystic ovary syndrome. Endocr Rev. 2015;36(5):487-525.
7. Goodarzi MO, Dumesic DA, Chazenbalk G, Azziz R. Polycystic ovary syndrome: etiology, pathogenesis and diagnosis. Nat Rev Endocrinol. 2011;7(4):219-231.
8. McCartney CR, Marshall JC. Polycystic ovary syndrome. N Engl J Med. 2016;375(1):54-64.
9. Rosner W, Auchus RJ, Azziz R, Sluss PM, Raff H. Position statement: utility, limitations, and

pitfalls in measuring testosterone: an Endocrine Society position statement. J Clin Endocrinol Metab. 2007;92(2):405-413.
10. Diamandis EP, Yu H. New biological functions of prostate-specific antigen? J Clin Endocrinol Metab. 1995;80(5):1515-1517.
11. Levesque M, Hu H, D'Costa M, Diamandis EP. Prostate-specific antigen expression by various tumors. J Clin Lab Anal. 1995;9(2):123-128.
12. Zarghami N, Grass L, Diamandis EP. Steroid hormone regulation of prostate-specific antigen gene expression in breast cancer. Br J Cancer. 1997;75(4):579-588.
13. Clements JA, Willemsen NM, Myers SA, Dong Y. The tissue kallikrein family of serine proteases: functional roles in human disease and potential as clinical biomarkers. Crit Rev Clin Lab Sci. 2004;41(3):265-312.
14. Gruschwitz MS, Breckwoldt M, Huber JC, Ludwig M, Pfleiderer A. Serum prostate-specific antigen levels in women with hirsutism or hyperandrogenism. Obstet Gynecol. 1995;85(5 Pt 1):760-763.
15. Yu H, Diamandis EP, Levesque M, et al. Prostate specific antigen in female body fluids. J Urol. 1995;153(6):2004-2008.
16. Oktem M, Gürbüz Y, Aksu K, Erbayraktar Z, Göktolga U, Yüce K. Serum prostate-specific antigen in women with polycystic ovary syndrome. Gynecol Endocrinol. 2004;19(5):263-267.
17. Gruschwitz MS, Hornstein OP, Benninghoff D, Stotz B. Immunoreactive prostate-specific antigen in female breast cancer and benign breast disease. Arch Gynecol Obstet. 1995;256(2):67-72.
18. Yu H, Diamandis EP, Monne M, Croce CM, Trapp T, Levesque MA. Oral contraceptives decrease prostate-specific antigen levels in female serum. Clin Biochem. 1998;31(3):193-197.
19. Diamandis EP, Yu H, Melegos DN. Prostate-specific antigen in female serum, a potential new marker of androgen excess. J Clin Endocrinol Metab. 1995;80(10):3009-3013.
20. Loy CJ, Evantash EG, Goebelsmann U, Stanczyk FZ. Serum prostate-specific antigen in women with polycystic ovary syndrome. Fertil Steril. 1998;70(3):535-539.
21. Sharma A, Tandon VR, Mahajan A, Sharma S. Serum prostate-specific antigen as a marker of hyperandrogenism in women with polycystic ovarian syndrome. JK Sci. 2018;20(2):68-72.
22. Deshmukh VL, Jadhav MV, Yelikar BR. Prostate specific antigen in polycystic ovarian syndrome. J Obstet Gynaecol India. 2020;70(3):214-219.
23. Zhang Y, Wang L, Liu X, et al. Serum prostate-specific antigen levels in women with polycystic ovary syndrome: a systematic review and meta-analysis. Reprod Biomed Online. 2019;39(4):611-619.
24. Riegman PH, Vlietstra RJ, van der Korput JA, Brinkmann AO, Trapman J. The promoter of the prostate-specific antigen gene contains a functional androgen responsive element. Mol Endocrinol. 1991;5(12):1921-1930.
25. Lilja H, Ulmert D, Vickers AJ. Prostate-specific antigen and prostate cancer: prediction, detection and monitoring. Nat Rev Cancer. 2008;8(4):268-278.
26. Ferguson KM, Reddy JK, Rao MS, Qian W, Engelhardt NJ, Clejan S. Prostate specific antigen in the female: immunohistochemical identification in normal and neoplastic tissues. Pathology. 1997;29(3):291-295.
27. Murtha PE, Zhu YS, Midzak A, et al. Characterization of KLK2 and PSA gene expression response to androgen in prostate cancer cells. Prostate. 2009;69(9):1045-1054.
28. Ehrmann DA, Barnes RB, Rosenfield RL. Polycystic ovary syndrome as a form of functional ovarian hyperandrogenism due to dysregulation of androgen secretion. Endocr Rev. 1995;16(3):322-353.
29. Azziz R, Carmina E, Dewailly D, 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.
30. Dunaif A. Insulin resistance and the polycystic ovary syndrome: mechanism and implications for pathogenesis. Endocr Rev. 1997;18(6):774-800.
31. Goodarzi MO, Jones MR, Li X, et al. Replication of association of DENND1A and THADA variants with polycystic ovary syndrome in European cohorts. J Med Genet. 2012;49(2):90-95.
32. Sturgeon CM, Duffy MJ, Stenman UH, et al. National Academy of Clinical Biochemistry laboratory medicine practice guidelines for use of tumor markers in testicular, prostate, colorectal, breast, and ovarian cancers. Clin Chem. 2008;54(12):e11-79.
33. Teede HJ, Misso ML, Costello MF, et al. Recommendations from the international evidence-based guideline for the assessment and management of polycystic ovary syndrome. Fertil Steril. 2018;110(3):364-379.