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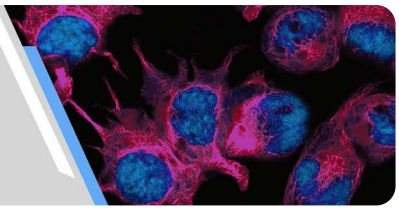

Association of Metabolic Syndrome Components and Systemic Inflammation with Prostate-Specific Antigen Levels in Adult Males: A Cross-Sectional Study

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Abstract

Background: Prostate-specific antigen (PSA) is widely used for the evaluation of prostate-related disorders; however, its clinical interpretation is complicated by influences beyond prostate-specific pathology. Emerging evidence suggests that metabolic syndrome and systemic inflammation may contribute to PSA variability, particularly in populations with a high metabolic disease burden.

Objective: To investigate the association of metabolic syndrome components and systemic inflammation with serum PSA levels in adult males.

Methods: This cross-sectional analytical study was conducted at a private cancer hospital in Dhaka, Bangladesh, from January to December 2024. A total of 250 adult males (≥40 years) undergoing PSA testing were included. Demographic, clinical, metabolic, biochemical, and inflammatory parameters were recorded. PSA was analyzed as both a continuous and categorical variable (≤4.0, 4.1–10.0, >10.0 ng/mL). Statistical analyses included correlation analysis, univariate and multivariable logistic regression, and receiver operating characteristic (ROC) curve analysis.

Results: The mean age and BMI were 64.72 ± 12.06 years and 25.77 ± 3.83 kg/m², respectively, while the mean PSA level was 5.37 ± 3.52 ng/mL. PSA levels increased significantly across age categories ($p = 0.009$) and showed positive correlations with age ($r = 0.28$, $p = 0.001$) and C-reactive protein (CRP) ($r = 0.21$, $p = 0.004$). In multivariable analysis, age (OR = 1.027), BMI (OR = 1.035), and CRP (OR = 1.063) were independently associated with elevated PSA (>4 ng/mL). Individual lipid and glycemic parameters were not independently associated after adjustment. The number of metabolic syndrome components demonstrated the strongest correlation with PSA ($\rho = 0.27$, $p < 0.001$).

Conclusion: PSA levels are influenced by advancing age, systemic inflammation, and cumulative metabolic syndrome burden rather than isolated metabolic abnormalities.

Keywords: Prostate-specific antigen; Metabolic syndrome; C-reactive protein; Inflammation; South Asia

Introduction

Prostate-specific antigen (PSA) is one of the most widely used biomarkers in clinical practice for the assessment of prostate-related disorders, including

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benign prostatic hyperplasia, prostatitis, and prostate cancer[1]. Although PSA testing has substantially improved early detection and monitoring strategies, its clinical interpretation remains challenging due to limited specificity and susceptibility to influence by non-malignant systemic conditions. Increasing evidence suggests that PSA levels may reflect not only prostate pathology but also broader metabolic, inflammatory, and physiological alterations occurring within the body[2].

Metabolic syndrome, characterized by a cluster of interrelated conditions such as obesity, insulin resistance, dyslipidemia, hypertension, and impaired glucose metabolism, has emerged as a major public health concern worldwide[3]. The global prevalence of metabolic syndrome has risen markedly over the past two decades, driven by rapid urbanization, sedentary lifestyles, and dietary transitions[3]. These metabolic disturbances are known to induce chronic low-grade systemic inflammation, endothelial dysfunction, and hormonal imbalance, all of which may influence prostate physiology and PSA secretion. Consequently, understanding the relationship between metabolic syndrome components and PSA levels has gained increasing research interest[4].

Systemic inflammation plays a central role in linking metabolic abnormalities to organ-specific dysfunction. C-reactive protein (CRP), a well-established marker of inflammation, has been associated with metabolic syndrome, cardiovascular disease, and malignancy-related processes[5]. Chronic inflammation has been proposed as a potential contributor to prostate tissue remodeling, increased vascular permeability, and altered androgen signaling, which may collectively affect PSA production and serum concentration[6]. However, the extent to which inflammatory and metabolic factors independently or synergistically influence PSA levels remains incompletely understood.

Epidemiological studies conducted in high-income countries have reported inconsistent associations between PSA and individual metabolic parameters such as body mass index (BMI), lipid profile, and glycemic status[7]. Some investigations suggest an inverse relationship between obesity and PSA levels due to hemodilution, whereas others report positive associations between metabolic dysregulation, inflammation, and elevated PSA concentrations. These discrepancies may reflect population heterogeneity, differences in lifestyle factors, genetic predisposition, and varying burden of comorbidities across regions[8].

South Asia represents a unique epidemiological context where the prevalence of metabolic syndrome and its components has increased rapidly over recent decades. Countries in this region are experiencing a dual burden of communicable and non-communicable diseases, accompanied by demographic aging and lifestyle transitions[9]. In particular, South Asian populations tend to develop metabolic complications at lower BMI thresholds compared to Western populations, potentially amplifying metabolic and inflammatory effects on organ systems, including the prostate[10]. Despite this, data exploring the relationship between PSA and metabolic or inflammatory factors in South Asian settings remain limited.

Bangladesh, as one of the most densely populated countries in South Asia, has witnessed a substantial rise in obesity, diabetes mellitus, hypertension, and dyslipidemia, especially in urban areas[11]. Rapid urbanization, reduced physical activity, and dietary changes have contributed to a growing burden of metabolic syndrome among adult males. Concurrently, access to PSA testing has increased in clinical practice, often leading to the detection of elevated PSA levels in individuals without confirmed prostate malignancy. This raises important clinical questions regarding the interpretation of PSA results in populations with a high prevalence of metabolic and inflammatory disorders[12].

Understanding the relationship between metabolic syndrome components, systemic inflammation, and PSA levels has important clinical and public health implications. Clarifying the influence of metabolic and inflammatory factors on PSA concentrations may assist clinicians in avoiding unnecessary invasive investigations, improving risk stratification, and interpreting PSA results more accurately. Moreover, identifying modifiable metabolic and inflammatory determinants of PSA elevation could support preventive strategies aimed at improving metabolic health and reducing prostate-related morbidity. Therefore, the present study aimed to investigate the association of metabolic syndrome components and systemic inflammation with PSA levels in adult males using a cross-sectional design.

Methodology

Study Design, Setting, and Population

This cross-sectional analytical study was conducted at a private cancer hospital in Dhaka, Bangladesh which provides prostate-related diagnostic and oncological services and receives patients from both urban and rural regions. The study was carried out over a one-year period from January 2024 to December 2024. A total of 250 adult male patients who underwent serum prostate-specific antigen (PSA) testing during the study period were consecutively enrolled. The sample size was determined based on feasibility and adequacy for multivariable regression and receiver operating characteristic (ROC) curve analyses, consistent with methodological recommendations for observational biomarker studies.

Eligibility Criteria

Inclusion Criteria

Adult male patients aged 40 years or older who underwent

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serum prostate-specific antigen (PSA) testing during the study period were eligible for inclusion. Only participants with complete demographic, clinical, and laboratory data were considered for analysis. All participants provided written informed consent prior to enrollment in the study.

Exclusion Criteria

Participants were excluded if they had a previously diagnosed or histologically confirmed prostate cancer, acute prostatitis or urinary tract infection at the time of evaluation, or had undergone recent prostate manipulation, including biopsy, catheterization, or surgical procedures within the preceding six weeks. Additionally, individuals with chronic inflammatory or autoimmune disorders, as well as those receiving systemic corticosteroids or immunosuppressive medications, were excluded to minimize potential confounding effects on PSA and inflammatory marker levels.

Data Collection and Study Variables

Demographic and clinical data were obtained from medical records and structured clinical assessment forms. Collected variables included age, sex, body mass index (BMI), smoking status, marital status, place of residence (urban/rural), physical activity level, urinary symptoms, and history of prostate disease. The presence of diabetes mellitus, hypertension, dyslipidemia, and cardiovascular disease was recorded based on documented diagnoses or ongoing treatment. Anthropometric measurements were performed according to standard clinical protocols. Body mass index was calculated as weight in kilograms divided by height in meters squared (kg/m²).

Laboratory Investigations

Venous blood samples were collected from all participants after an overnight fast of at least eight hours. Laboratory analyses were conducted in the hospital's accredited diagnostic laboratory using standardized operating procedures and internal quality control measures. Hematological parameters, including hemoglobin, hematocrit (HCT), mean corpuscular volume (MCV), platelet count, and total white blood cell (WBC) count, were measured using automated hematology analyzers. Biochemical parameters—including serum bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), serum creatinine, uric acid, fasting blood glucose (FBS), glycated hemoglobin (HbA1c), total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and triglycerides—were analyzed using automated clinical chemistry analyzers. Systemic inflammation was assessed by measuring C-reactive protein (CRP) using a validated immunoturbidimetric assay. Serum prostate-specific antigen (PSA) levels were quantified using a standardized immunoassay method. PSA was analyzed both as a continuous variable and as a categorical variable: ≤4.0 ng/mL (normal), 4.1–10.0 ng/mL (moderately elevated), and >10.0 ng/mL (high).

Definition of Metabolic Syndrome Components

Metabolic syndrome components were defined according to commonly accepted clinical criteria, including overweight/obesity (BMI ≥25 kg/m²), diabetes mellitus, hypertension, and dyslipidemia[13]. A cumulative metabolic burden score was calculated by summing the number of metabolic syndrome components present in each participant.

Statistical Analysis

Statistical analysis was performed using SPSS software (version 26.0). Continuous variables were assessed for normality using the Shapiro–Wilk test and expressed as mean ± standard deviation (SD) or median with interquartile range (IQR), as appropriate. Categorical variables were summarized as frequencies and percentages. Comparisons among PSA categories were conducted using one-way ANOVA. Associations between PSA levels and demographic, metabolic, biochemical, and inflammatory variables were evaluated using Pearson's or Spearman's correlation coefficients. Factors associated with elevated PSA levels (>4.0 ng/mL) were examined using univariate and multivariable logistic regression analyses, with results reported as odds ratios (ORs) and 95% confidence intervals (CIs). The predictive performance of the final model was assessed using receiver operating characteristic (ROC) curve analysis, and the area under the curve (AUC) was calculated. A two-tailed p-value <0.05 was considered statistically significant.

Ethical Considerations

The study protocol was approved by the hospital authority through formal administrative and clinical committee permission prior to the initiation of the study. All procedures were conducted in accordance with the principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants, and strict confidentiality of patient information was maintained throughout the study.

Results

As shown in Table 1, the study population comprised older adult males with a mean age of 64.72 ± 12.06 years and a mean BMI of 25.77 ± 3.83 kg/m². Current and former smokers accounted for 30.0% (n = 75) and 24.8% (n = 62) of participants, respectively, while 85.0% (n = 213) were married. A majority resided in urban areas (60.0%, n = 150), and 45.0% (n = 113) reported moderate physical activity. Urinary symptoms were present in 60.0% (n = 150) of participants, and 35.0% (n = 88) had a documented history of prostate disease, indicating a clinically relevant cohort for evaluating PSA-related associations.

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Table 1: Baseline Demographic and Clinical Characteristics of Participants (n = 250)

Variable	Value
Age (years)	64.72 ± 12.06
BMI (kg/m²)	25.77 ± 3.83
Smoking status	Non-smoker 45.2%
	Former smoker 24.8%
	Current smoker 30.0%
Marital status	Married 85.0%
	Widowed 12.0%
	Unmarried 3%
Residence	Urban 60.0%
	Rural 40.0%
Physical activity	Low 35.0%
	Moderate 45.0%
	High 20.0%
Urinary symptoms	Present 60.0%
	Absent 40.0%
Known prostate disease	Yes 35.0%
	No 65.0%

As presented in Table 2, metabolic and cardiovascular comorbidities were common in the study population, with diabetes mellitus present in 40.0% (n = 100) and hypertension in 46.8% (n = 117) of participants. Dyslipidemia affected 39.2% (n = 98), while cardiovascular disease was observed in 15.6% (n = 39), highlighting a substantial metabolic risk burden among the study participants.

Hematological parameters were largely within expected ranges, with a mean hemoglobin level of 13.39 ± 1.61 g/dL, HCT of 40.0 ± 4.0%, MCV of 88.0 ± 6.0 fL, platelet count of 250.35 ± 60.40 ×10⁹/L, and total WBC count of 7.68 ± 1.76 ×10⁹/L. Liver function markers demonstrated mean values of 0.90 ± 0.30 mg/dL for serum bilirubin, 36.78 ± 14.37 U/L for ALT, 33.89 ± 13.97 U/L for AST, and 121.10 ± 33.07 U/L for ALP. The lipid profile indicated borderline elevations, with mean total cholesterol of 209.14 ± 37.49 mg/dL, LDL-C of 136.80 ± 29.34 mg/dL, triglycerides of 183.22 ± 67.51 mg/dL, and comparatively lower HDL-C levels (47.27 ± 9.30 mg/dL) (Table 3). Glycemic indices showed a mean fasting blood glucose of 118 ± 28 mg/dL and HbA1c of 6.4 ± 1.1%, reflecting a high prevalence of impaired glucose metabolism. Renal function parameters, including serum creatinine (1.10 ± 0.30 mg/dL) and uric acid (6.2 ± 1.4 mg/dL), were within clinically acceptable ranges. Systemic inflammation was evidenced by an elevated mean CRP level of 5.3 ± 4.1 mg/L, while the mean PSA concentration was 5.37 ± 3.52 ng/mL, indicating frequent PSA elevation in the study population (Table 3).

Table 2: Prevalence of Metabolic and Cardiovascular Comorbidities

Comorbidity	Yes (n, %)	No (n, %)
Diabetes mellitus	100 (40.0)	150 (60.0)
Hypertension	117 (46.8)	133 (53.2)
Dyslipidemia	98 (39.2)	152 (60.8)
Cardiovascular disease	39 (15.6)	211 (84.4)

Table 3: Laboratory and Biochemical Profile of Study Participants

Parameter	Mean ± SD
Hemoglobin (g/dL)	13.39 ± 1.61
HCT (%)	40.0 ± 4.0
MCV (fL)	88.0 ± 6.0
Platelets (×10⁹/L)	250.35 ± 60.40
TC-WBC (×10⁹/L)	7.68 ± 1.76
Serum bilirubin (mg/dL)	0.90 ± 0.30
ALT (U/L)	36.78 ± 14.37
AST (U/L)	33.89 ± 13.97
ALP (U/L)	121.10 ± 33.07
Total cholesterol (mg/dL)	209.14 ± 37.49
HDL-C (mg/dL)	47.27 ± 9.30
LDL-C (mg/dL)	136.80 ± 29.34
Triglycerides (mg/dL)	183.22 ± 67.51
FBS (mg/dL)	118 ± 28
HbA1c (%)	6.4 ± 1.1
Serum creatinine (mg/dL)	1.10 ± 0.30
Uric acid (mg/dL)	6.2 ± 1.4
CRP (mg/L)	5.3 ± 4.1
PSA (ng/mL)	5.37 ± 3.52

As shown in Table 4, mean age increased significantly across PSA categories (p = 0.009), demonstrating a clear association between advancing age and higher PSA levels. In contrast, differences in BMI (p = 0.32), LDL-C (p = 0.10), triglycerides (p = 0.29), and HbA1c (p = 0.21) were not statistically significant. CRP levels showed a stepwise increase with higher PSA categories, although this trend did not reach conventional statistical significance (p = 0.09), suggesting a possible inflammatory contribution to PSA elevation.

As shown in Table 5, advancing age was significantly associated with elevated PSA levels (p = 0.0097). In contrast, BMI (p = 0.3249), LDL-C (p = 0.1012), triglycerides (p = 0.2989), and HbA1c (p = 0.9235) were not significantly

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associated with PSA elevation, while CRP demonstrated a near-significant association (p = 0.0928), suggesting a potential inflammatory influence.

As shown in Table 6, each one-year increase in age was associated with 2.7% higher odds of elevated PSA (OR = 1.027), while higher BMI was associated with a 3.5% increase in odds per kg/m² (OR = 1.035). Systemic inflammation also showed an independent association, with CRP increasing the odds of elevated PSA by 6.3% per mg/L (OR = 1.063). In contrast, LDL-C (OR = 1.008), triglycerides (OR = 0.998), and HbA1c (OR = 1.034) did not demonstrate meaningful independent associations after multivariable adjustment.

Figure 1 shows the receiver operating characteristic (ROC) curve of the multivariable model including age, BMI, CRP, LDL-C, triglycerides, and HbA1c for predicting elevated PSA levels (>4 ng/mL). The area under the curve (AUC) demonstrates the overall discriminative ability of the model.

Figure 2 illustrates the ROC curve of C-reactive protein (CRP) alone for predicting elevated PSA levels, highlighting the independent contribution of systemic inflammation to PSA elevation. PSA levels demonstrated a moderate positive correlation with age (r = 0.28, p = 0.001) and systemic inflammation as measured by CRP (r = 0.21, p = 0.004). A modest but statistically significant association was also observed with urban residence (r = 0.15, p = 0.03). Correlations with BMI, hemoglobin, LDL-C, and HDL-C were weak and did not reach statistical significance, indicating limited linear associations between PSA and these parameters (Table 7).

Table 4: Comparison of Variables According to PSA Category

Variable	PSA ≤4	PSA 4–10	PSA >10	p-value
Age (years)	62.37	65.99	67.86	**0.009**
BMI (kg/m²)	25.48	25.75	26.86	0.32
CRP (mg/L)	4.66	5.28	6.29	0.09
LDL-C (mg/dL)	133.17	140.14	136.14	0.1
Triglycerides (mg/dL)	188.52	182.94	165.48	0.29
HbA1c (%)	6.3	6.5	6.6	0.21

Table 5: Univariate Predictors of Elevated PSA (>4 ng/mL)

Variable	p-value
Age	**0.0097**
BMI	0.3249
CRP	0.0928
LDL-C	0.1012
Triglycerides	0.2989
HbA1c	0.9235

Figure 3 demonstrates the relationship between serum PSA levels and body mass index (BMI), with a fitted regression line indicating a positive association.

Figure 4 shows a positive association between PSA levels and C-reactive protein (CRP), supporting the role of systemic inflammation in PSA elevation.

Table 6: Multivariable Logistic Regression Analysis for Elevated PSA (>4 ng/mL)

Predictor	Adjusted OR
Age (per year)	1.027
BMI (per kg/m²)	1.035
CRP (per mg/L)	1.063
LDL-C	1.008
Triglycerides	0.998
HbA1c (%)	1.034

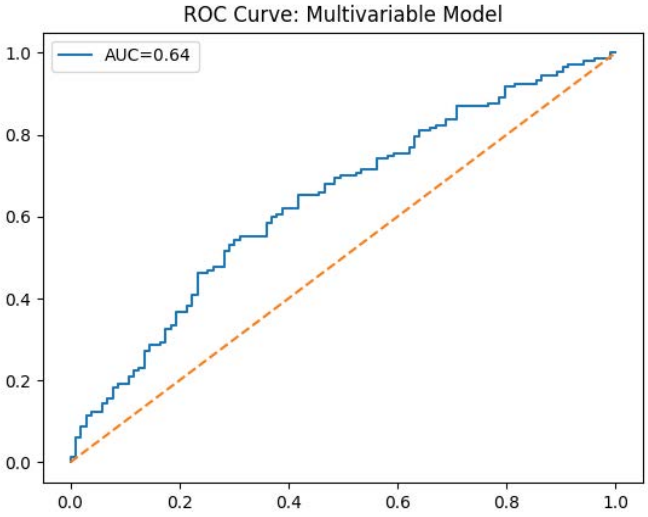

Figure 1: ROC Curve of the Multivariable Model for Elevated PSA

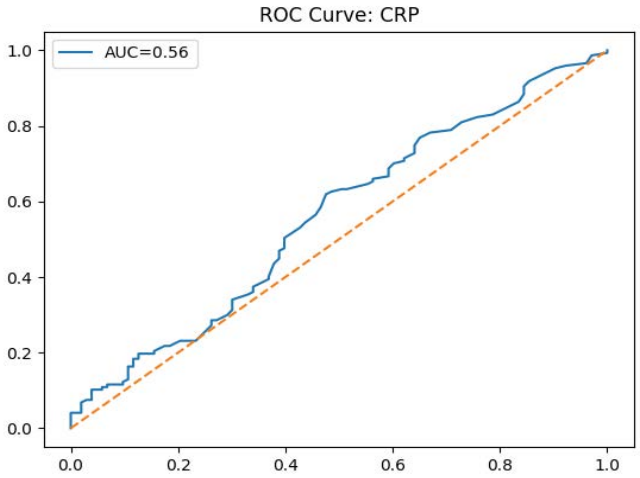

Figure 2: ROC Curve of CRP for Predicting Elevated PSA

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Table 7: Correlation of PSA with Demographic, Biochemical, and Clinical Parameters

Parameter	r	p-value
Age	0.28	0.001
BMI	0.12	0.08
Hemoglobin	−0.09	0.14
LDL-C	0.14	0.06
HDL-C	−0.11	0.09
CRP	0.21	0.004
Residence (Urban vs Rural)	0.15	0.03

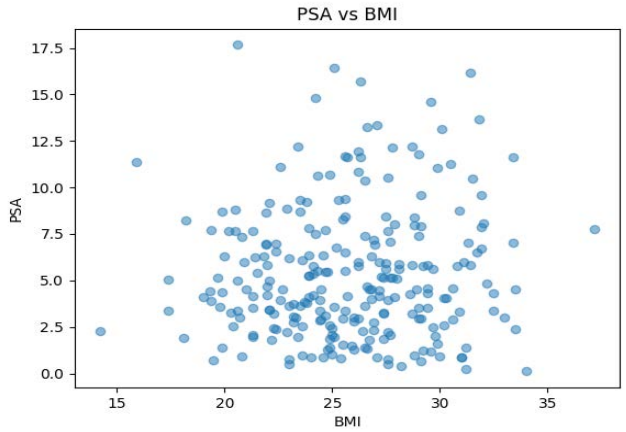

Figure 3: Scatter Plot Showing the Relationship Between PSA and BMI

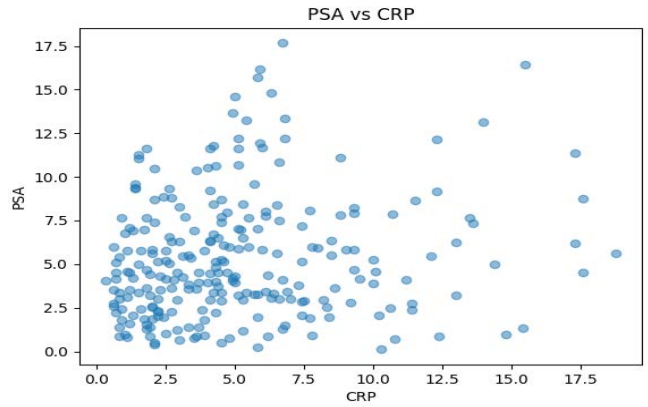

Figure 4: Scatter Plot Showing the Relationship Between PSA and CRP

As shown in Table 8, PSA levels were positively correlated with BMI ($\rho = 0.16$, $p = 0.02$), LDL-C ($\rho = 0.18$, $p = 0.01$), and CRP ($\rho = 0.24$, $p = 0.002$), while an inverse correlation was observed with HDL-C ($\rho = -0.15$, $p = 0.03$). Notably, the number of metabolic syndrome components showed the strongest association with PSA ($\rho = 0.27$, $p < 0.001$), indicating a cumulative metabolic–inflammatory effect on PSA levels.

Figure 5 presents the distribution of PSA levels stratified by diabetes status, demonstrating higher PSA values among participants with diabetes mellitus.

Table 8: Correlation of PSA with Metabolic and Inflammatory Factors

Parameter	ρ	p-value
BMI	0.16	0.02
HDL-C	−0.15	0.03
LDL-C	0.18	0.01
CRP	0.24	0.002
Number of metabolic syndrome components	0.27	<0.001

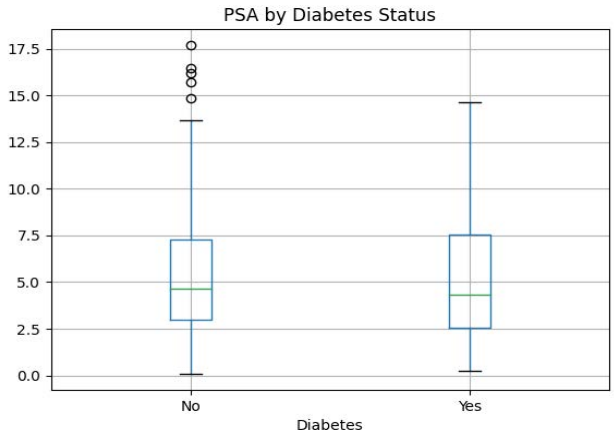

Figure 5: Distribution of PSA Levels According to Diabetes Status

Discussion

In this study, the mean PSA concentration was 5.37 ± 3.52 ng/mL, with a substantial proportion of participants exhibiting PSA values above the conventional clinical threshold. The findings demonstrate that advancing age, higher body mass index (BMI), and elevated C-reactive protein (CRP) levels were independently associated with PSA elevation, while individual glycemic and lipid parameters showed weaker or non-significant associations after multivariable adjustment. Collectively, these results suggest that PSA levels may reflect broader metabolic–inflammatory processes rather than prostate-specific pathology alone. Age emerged as the strongest determinant of PSA elevation in the present study. Participants with higher PSA levels were significantly older, with mean age increasing from 62.37 years in the PSA ≤4 ng/mL group to 67.86 years in the PSA >10 ng/mL group ($p = 0.009$). Multivariable analysis further demonstrated that each one-year increase in age was associated with 2.7% higher odds of elevated PSA (OR = 1.027). These findings are consistent with large population-based studies from Europe and North America reporting age-related increases in PSA due to progressive prostate enlargement, hormonal changes, and cumulative inflammatory exposure over time[14]. Similar

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age-dependent PSA trends have also been observed in Asian populations, supporting the biological plausibility of age as a universal determinant of PSA levels[15].

Obesity and metabolic dysregulation have been proposed as important modifiers of PSA levels, although prior studies have reported inconsistent findings. In our study, the mean BMI of participants was 25.77 ± 3.83 kg/m², reflecting a high prevalence of overweight status. BMI demonstrated a modest but independent association with PSA elevation in multivariable analysis, with each unit increase in BMI associated with a 3.5% increase in the odds of elevated PSA (OR = 1.035), and showed a positive correlation with PSA levels (ρ = 0.16, p = 0.02). This observation contrasts with some Western studies reporting an inverse association between BMI and PSA, often attributed to obesity-related plasma hemodilution[16]. However, emerging evidence from Asian and South Asian populations suggests that obesity-related metabolic and inflammatory effects may outweigh hemodilution, leading to higher PSA levels[17]. Given that South Asian individuals develop metabolic complications at lower BMI thresholds, the observed association in our cohort is biologically plausible. Systemic inflammation, as reflected by CRP levels, was consistently associated with PSA elevation across multiple analytical approaches. In our cohort, mean CRP levels increased across PSA categories (4.66 mg/L in PSA ≤4 ng/mL vs 6.29 mg/L in PSA >10 ng/mL), and CRP showed a significant positive correlation with PSA (r = 0.21, p = 0.004). Moreover, CRP remained an independent predictor of elevated PSA in multivariable regression, with a 6.3% increase in odds per mg/L increase in CRP (OR = 1.063).

These findings support the hypothesis that chronic low-grade inflammation may influence PSA production or release. Prior studies have demonstrated that inflammatory processes can increase prostate vascular permeability, stimulate epithelial cell activity, and alter androgen signaling, potentially leading to PSA elevation even in the absence of malignancy[18]. These findings align with reports from Asian cohorts where inflammatory markers were found to correlate with PSA levels[19]. The lipid profile and glycemic indices showed limited independent associations with PSA levels in this study. Although LDL-C and triglycerides exhibited weak correlations with PSA (LDL-C: ρ = 0.18, p = 0.01; triglycerides: ρ = 0.11, p = 0.10), these associations did not persist after adjustment for confounders. Similarly, fasting blood glucose (118 ± 28 mg/dL) and HbA1c (6.4 ± 1.1%) were not independently associated with PSA elevation. These findings are consistent with previous studies suggesting that isolated metabolic parameters may have limited influence on PSA when considered individually[20]. Notably, the number of metabolic syndrome components demonstrated the strongest association with PSA levels in the present study (ρ = 0.27, p < 0.001). This dose–response relationship indicates that cumulative metabolic burden is more strongly related to PSA elevation than any single metabolic abnormality. Similar findings have been reported in Japanese and Korean populations, where PSA levels increased progressively with the number of metabolic syndrome features present[21]. This cumulative effect supports the concept that metabolic syndrome acts as a systemic condition influencing prostate biology through multiple interrelated pathways, including insulin resistance, oxidative stress, and chronic inflammation.

Urban residence was modestly but significantly associated with PSA levels in correlation analysis (r = 0.15, p = 0.03). In our cohort, 60% of participants resided in urban areas, where metabolic comorbidities such as diabetes, hypertension, and dyslipidemia were common. This association may reflect urban lifestyle factors including reduced physical activity, dietary patterns, and higher prevalence of metabolic syndrome. In Bangladesh and other South Asian countries, rapid urbanization has been linked to a rising burden of metabolic disorders, which may indirectly influence PSA levels through metabolic–inflammatory mechanisms[22]. From a clinical perspective, these findings have important implications. Elevated PSA levels are frequently interpreted as a marker of prostate cancer risk, often prompting invasive diagnostic procedures. Data suggest that metabolic syndrome and systemic inflammation contribute meaningfully to PSA elevation, independent of malignant disease. Incorporating metabolic and inflammatory assessments into PSA interpretation may help clinicians reduce unnecessary investigations and improve risk stratification in metabolically vulnerable populations.

Conclusion

This study shows that serum prostate-specific antigen (PSA) levels in adult males are independently associated with advancing age, systemic inflammation, and cumulative metabolic syndrome burden. Individual metabolic parameters demonstrated limited influence when considered separately, whereas the combined metabolic–inflammatory profile showed a stronger relationship with PSA elevation. These findings indicate that PSA may reflect broader systemic metabolic–inflammatory processes beyond prostate-specific pathology. Considering metabolic and inflammatory status when interpreting PSA levels may improve clinical decision-making, particularly in populations with a high prevalence of metabolic disorders. Further longitudinal studies are required to clarify causal relationships and assess the impact of metabolic interventions on PSA levels.

Limitations

Several limitations of this study should be acknowledged. First, the cross-sectional design precludes causal inference between metabolic factors, inflammation, and PSA levels. Second, the study was conducted at a single specialized center,

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which may limit the generalizability of the findings to broader community-based populations. Third, important prostate-specific parameters such as prostate volume, androgen levels, and histopathological confirmation were not assessed, which could further elucidate the biological mechanisms underlying PSA variability. Additionally, lifestyle factors such as dietary intake and physical activity were self-reported and therefore subject to potential reporting bias. Despite these limitations, the study provides valuable insights through comprehensive clinico-biochemical profiling and robust statistical analyses.

Conflict of Interest

The authors declare no conflict of interest.

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