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RESEARCH

Prostate-Specific Antigen and Cardio-Metabolic Biomarkers in Nigerian Men With and Without Type 2 Diabetes: A Case-Control Study

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Abstract

Background Prostate-specific antigen (PSA) is influenced by abnormalities commonly seen in type 2 diabetes, yet evidence from African populations remains limited. Understanding how PSA relates to cardio-metabolic biomarkers in Nigerian men may improve prostate health assessment in settings with increasing rates of diabetes and obesity.

Objectives This study compared PSA concentrations and cardio-metabolic biomarkers between men with type 2 diabetes and non-diabetic controls, examined age-related variation, and assessed associations involving body mass index, lipid profile, blood pressure, and duration of diabetes.

Methods A case-control study was conducted among 86 diabetic men and 60 non-diabetic controls attending two tertiary hospitals in Nigeria. Anthropometric indices and blood pressure were measured. Fasting glucose, total PSA, free PSA, and lipid fractions were analysed using standard biochemical assays. Statistical analyses included t tests, chi-square tests, one-way analysis of variance, Spearman correlation, and multivariable linear regression to adjust for confounding.

Results Diabetic men represented 58.9% of the sample and were significantly older than controls, reflecting the pattern of clinic attendance during the recruitment period ($p<0.001$). Total PSA did not differ significantly between diabetic and non-diabetic participants ($t=1.65$, $p=0.10$), whereas free PSA was higher among diabetic men ($t=2.19$, $p=0.03$). Diabetic men had significantly higher BMI ($t=4.49$, $p<0.001$), fasting glucose ($t=2.70$, $p<0.01$), systolic blood pressure ($t=3.81$, $p<0.001$), and diastolic blood pressure ($t=5.88$, $p<0.001$). Significant differences were also found for total cholesterol ($p=0.03$), HDL cholesterol ($p<0.001$), and LDL cholesterol ($p<0.01$). In adjusted analyses, diabetes status was not independently associated with total PSA, whereas age remained an important predictor of PSA variation. The association between diabetes status and free PSA was attenuated after adjustment. Duration of diabetes was not associated with PSA or glucose.

Conclusion PSA interpretation in Nigerian men should take account of age, adiposity, and broader cardio-metabolic status. The observed difference in free PSA warrants further investigation but should be interpreted cautiously. These findings highlight the importance of considering metabolic context when evaluating prostate health in men with diabetes.

Keywords Type 2 diabetes mellitus · Prostate-specific antigen · Dyslipidaemia · Fasting blood glucose · Body mass index · Cardio-metabolic biomarkers · Nigeria

Introduction

Prostate-specific antigen (PSA) is widely used in the assessment of prostate health and remains the principal biochemical marker for detecting early prostate abnormalities in men [1]. PSA concentrations increase with age because prostate volume grows steadily across the life course, which makes age an important determinant when interpreting PSA values in clinical and research settings [2]. Age related differences have also been observed in the prevalence of metabolic disorders such as obesity, hypertension, dyslipidaemia, and impaired glucose regulation, all of which form the cluster of metabolic markers typically associated with type 2 diabetes [3, 4]. These features are strongly influenced by ageing and contribute to biological pathways that affect prostate growth, inflammation, and endocrine signalling [5]. Understanding how these markers relate to PSA is therefore

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clinically relevant, particularly in settings where metabolic diseases are increasing rapidly.

The global prevalence of type 2 diabetes has risen sharply over the past two decades, with recent estimates suggesting that more than 500 million adults now live with the condition and the total number of individuals with diabetes is projected to rise to 853 million by 2050 [6]. Nigeria has one of the highest burdens in Africa, and studies consistently report a growing prevalence in the southeastern region. Diabetes has been linked to changes in lipid metabolism, insulin resistance, systemic inflammation, and altered androgen levels, all of which may influence PSA values [7, 8]. Evidence has also suggested that men with type 2 diabetes may have lower PSA levels than non-diabetic men, although findings are inconsistent across populations [9]. Possible explanations include diabetes related endocrine changes and the effect of obesity on plasma volume and PSA dilution [10]. These uncertainties highlight the need for studies that examine PSA and cardio-metabolic markers together.

Cardio-metabolic markers such as body mass index, blood pressure, fasting plasma glucose, and lipid fractions reflect interconnected pathways involving metabolic regulation, vascular function, and systemic inflammation [11]. Dyslipidaemia, elevated triglycerides, and lower high-density lipoprotein (HDL) cholesterol are common features of the metabolic disturbances observed in type 2 diabetes and have been associated with increased risk of prostate abnormalities in some studies [12]. Hypertension and increased central adiposity are also more common in older adults and may contribute to prostate enlargement and hormonal alterations that influence PSA secretion [13]. Because these biomarkers share common physiological pathways with PSA, it is important to assess how they differ between diabetic and non-diabetic men.

Age plays a central role in the interpretation of both PSA and metabolic biomarkers. PSA values rise with age even in healthy men, and metabolic risk increases substantially from midlife onwards [13, 14]. Age stratification is therefore necessary for examining these relationships. In addition, the duration of diabetes may influence metabolic status and systemic inflammation, yet its association with PSA remains unclear. Previous studies have reported mixed findings, with some suggesting no relationship and others reporting modest associations [15]. More evidence is needed, particularly in African populations where biological, lifestyle, and healthcare factors may differ.

Obesity is another potential modifier of metabolic and prostate related outcomes. Higher body mass index (BMI) is strongly associated with dyslipidaemia, elevated blood pressure, and impaired glucose regulation [14, 16]. Obesity may also lower PSA concentrations through haemodilution or endocrine mechanisms that reduce androgen production [17, 18]. Examining BMI categories in relation to lipid profile, PSA, and blood pressure can therefore provide a clearer understanding of how adiposity shapes metabolic and prostate health in men with and without diabetes.

Despite the clinical importance of these relationships, limited research has examined PSA alongside a full cardio-metabolic profile in Nigerian men, and few studies have explored these associations across age groups [9, 10]. Understanding these patterns may support more accurate interpretation of PSA in populations with a rising prevalence of diabetes and obesity. This study compared PSA concentrations and cardio-metabolic biomarkers between men with type 2 diabetes and non-diabetic controls, examined age-related variation, and assessed associations involving body mass index, lipid profile, blood pressure, and duration of diabetes.

Materials and Methods

Area of Study

The study was conducted in two tertiary hospitals in Enugu State in Southeast Nigeria. According to the National Bureau of Statistics [19], Enugu State had a population of 3,267,837 persons in the 2006 census and was projected to be approximately 4,690,100 persons in 2022. These were the University of Nigeria Teaching Hospital Ituku-Ozalla and the Enugu State University Teaching Hospital. Both facilities run established diabetes and general medical outpatient clinics and have chemical pathology laboratories capable of performing prostate specific antigen testing and full metabolic profiling. These hospitals were selected because they provide routine care for a broad demographic of adult men and offer the infrastructure needed for sample handling and laboratory analysis.

Inclusion Criteria and Exclusion Criteria

Eligible participants were men aged 40 to 75 years. The diabetic group consisted of men with a documented diagnosis of type 2 diabetes mellitus confirmed in their medical records. Controls were men in the same age range attending outpatient clinics for routine care who had no previous diagnosis of diabetes, no symptoms suggestive of hyperglycaemia, and fasting plasma glucose within the non-diabetic range.

Participants taking lipid-lowering agents, 5-alpha reductase inhibitors, or other medications known to influence PSA, glucose, or lipid metabolism were excluded. Medication history was verified through patient records and direct confirmation during the clinic visit. However, documentation was occasionally incomplete, and residual uncertainty

regarding prior statin or antihypertensive use cannot be entirely ruled out.

Participants with known prostate cancer or documented benign prostatic hyperplasia were excluded where such diagnoses were recorded in clinical notes. However, routine screening for subclinical prostate conditions was not feasible within the study setting. In addition, factors known to influence PSA levels, including recent ejaculation, digital rectal examination, urogenital instrumentation, and prolonged cycling, were not systematically controlled prior to sample collection because of the pragmatic nature of clinic-based recruitment. These factors are acknowledged as potential sources of variability.

Ethical Consideration and Consent to Participate in the Study

The study protocol was approved by the College of Medicine Research and Ethics Committee, University of Nigeria Enugu campus (Reference: 073/07/2019). All procedures were conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrolment. Study identification numbers were used to maintain anonymity and protect confidentiality.

Research Design and Sample Size Calculation

A case-control design was utilised in this study. Sample size was determined using a single proportion formula [20] based on a reported prevalence of type 2 diabetes of 4.6% in Southeast Nigeria. Using a 95% confidence interval and a precision of 5%, the minimum required sample for the diabetic group was 67.4 participants. This was increased by 10% to allow for non-response, giving a target of 86 men. The same recruitment target was planned for controls; however, 60 eligible men met the inclusion criteria and completed all procedures during the study period. All analyses were conducted on the final sample of 146 participants.

Participants and Recruitment

Participants were recruited consecutively during routine outpatient visits. Research assistants screened clinic attendees using the eligibility criteria and confirmed diabetes status or absence of diabetes through medical records and fasting plasma glucose. Details of the study were explained, and written consent was obtained. Consecutive sampling ensured that all eligible men presenting during recruitment were invited to participate. This approach was practical in the clinical setting and allowed consistent application of the inclusion and exclusion criteria.

Determination of Body Mass Index and Blood Pressure

Anthropometric measurements followed standard procedures. Height was measured using a stadiometer to the nearest 0.1 cm. Weight was recorded with a calibrated analogue scale to the nearest 0.1 kg. Body mass index was calculated as weight in kilogrammes divided by height in metres squared, and categories were assigned using the World Obesity Federation classification [21]. Blood pressure was measured twice during a single clinic visit using a manual sphygmomanometer after the participant had rested for at least five minutes in a seated position. The average of the two readings was used for analysis. Hypertension was defined as systolic blood pressure of 140 mmHg or higher and diastolic blood pressure of 90 mmHg or higher according to International Society of Hypertension guidelines [22].

Blood Sample Collection and Processing

A fasting venous blood sample was collected after an overnight fast of at least eight hours. Two millilitres were placed in a sodium fluoride-potassium oxalate tube for plasma glucose analysis. Three millilitres were placed in a plain tube for serum extraction. Samples were centrifuged at the recommended speed and temperature for serum and plasma separation. Plasma and serum were stored at four degrees Celsius and analysed within the allowable stability period. All reagents and consumables were used according to manufacturer instructions.

Biochemical Assays and Quality Control

Biochemical analyses were conducted in the chemical pathology laboratories of the two hospitals by trained personnel. Plasma glucose was analysed using the glucose oxidase method. Serum total cholesterol, HDL cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides, and very-low-density lipoprotein (VLDL) cholesterol were measured using standard enzymatic colorimetric assays. Total and free PSA were determined using a sandwich enzyme linked immunosorbent assay. Quality control procedures included daily calibration of analytical instruments and the use of commercial control materials at low, medium, and high concentration ranges during each assay run. Control values were reviewed before processing participant samples. Runs with out-of-range control values were repeated after recalibration. The laboratories participate in routine internal quality assurance, which ensured consistency and reliability of all assay results.

Determination of Prostate Specific Antigen

Serum PSA was measured according to the manufacturer's protocol. Concentrations were determined from calibration standards included with the assay kit. A value of 4 ng/mL or lower was considered within the accepted reference range for total PSA in this study [23]. Free PSA was also assessed and a value of 1.26 ng/mL or less was considered normal [24]. Although PSA interpretation is age dependent, age adjusted ranges were not available across all clinical records. The fixed threshold was therefore applied consistently during biochemical classification, and age effects were examined separately in the statistical analysis.

Determination of Lipid Profile

Total cholesterol was measured using an enzymatic method based on cholesterol esterase, cholesterol oxidase, and peroxidase. HDL cholesterol was analysed following selective precipitation of non-HDL lipoproteins and subsequent enzymatic quantification. Triglycerides were measured using an enzymatic assay involving lipase-mediated hydrolysis and chromogenic detection. LDL cholesterol was calculated using the Friedewald [25] equation for samples with triglyceride levels below the limit for reliable calculation. Lipid results were interpreted using established reference ranges.

Statistical Analysis

Analysis was performed using SPSS version 22. Descriptive statistics summarised demographic and clinical characteristics using means with standard deviations for continuous variables and frequencies with percentages for categorical variables. Independent samples t tests were used to compare PSA, body mass index, blood pressure, fasting glucose, and lipid profile values between diabetic and control groups. Chi-square tests were used for categorical variables including age group, BMI categories, PSA classification, and lipid categories. One-way analysis of variance examined age-group differences in PSA, fasting glucose, and blood pressure within diabetic and control groups. Post hoc tests were performed where significant effects were detected. Spearman correlation analysis was used to assess associations between duration of diabetes and PSA or fasting glucose.

Normality and homogeneity of variance were assessed before applying parametric tests. Because fasting glucose showed a markedly skewed distribution, values were checked for data entry errors and log-transformed for inferential analyses where appropriate. Analyses used complete-case data. Statistical significance was set at $p<0.05$. To address potential confounding, multivariable linear regression analyses were performed to examine the association between diabetes status and PSA outcomes while adjusting for age, body mass index, and selected cardio-metabolic variables. Regression coefficients, 95% confidence intervals, and p values were estimated, and model assumptions were checked before interpretation.

Results

Participant Characteristics

A total of 146 men participated in the study, including 86 men with type 2 diabetes and 60 non-diabetic controls. Table 1 presents their sociodemographic characteristics. There were marked differences in age distribution. Most participants with diabetes were aged 60 years or older, while the majority of controls were younger than 60 years. A chi-square test confirmed that age distribution differed significantly between groups ($p<0.001$). Because PSA and several cardio-metabolic markers vary with age, this imbalance was considered in the interpretation of between-group

Table 1 Sociodemographic characteristics of participants ($n=146$)

Variable	Diabetic ($n=86$)	Control ($n=60$)
Age group (years)		
40–49	14 (16.3)	36 (60.0)
50–59	12 (14.0)	18 (30.0)
60–69	48 (55.8)	4 (6.7)
70–79	12 (14.0)	2 (3.3)
Marital status		
Married	84 (97.7)	60 (100.0)
Single	2 (2.3)	0 (0.0)
Occupation		
Business or self-employed	46 (53.5)	20 (33.3)
Civil servant	10 (11.6)	36 (60.0)
Artisan	6 (7.0)	0 (0.0)
Clergy	2 (2.3)	2 (3.3)
Retired	16 (18.6)	0 (0.0)
Education level		
Tertiary	34 (39.5)	50 (83.3)
Secondary	18 (20.9)	10 (16.7)
Primary	32 (37.2)	0 (0.0)
Smoking status		
Non-smoker	76 (88.4)	58 (96.7)
Current	2 (2.3)	0 (0.0)
Ex-smoker	6 (7.0)	0 (0.0)
Alcohol intake		
None	26 (30.2)	58 (96.7)
Regular	51 (59.3)	0 (0.0)
Occasional	9 (10.5)	2 (3.3)

Note: Differences in age-group distribution between groups were statistically significant ($\chi^2 = 45.9$, $p<0.001$). Regular alcohol intake was defined as consumption of 3–4 pints of alcoholic drinks about 3–6 times per week. Occasional alcohol intake was defined as consumption of 1–2 pints less than twice per month

comparisons and in adjusted regression analyses. Other demographic characteristics, including marital status, occupation, education level, smoking, and alcohol use, varied across groups. This age imbalance was taken into account in the interpretation of between-group comparisons and in adjusted regression analyses.

Comparison of PSA Levels Between Diabetic and Non-Diabetic Men

Independent samples t tests showed that total PSA levels did not differ significantly between the diabetic and control groups (t = 1.65, $p = 0.10$). Free PSA was significantly higher among men with diabetes (t = 2.19, $p = 0.03$). When PSA values were classified according to conventional clinical thresholds, chi-square testing did not indicate a significant association between PSA category and diabetes status. Because PSA is age dependent and age distribution differed significantly between groups ($p < 0.001$), adjusted analyses were performed to examine whether these associations persisted after accounting for potential confounding. Table 2 summarises the unadjusted group comparisons, while Fig. 1 illustrates mean total PSA across age categories.

Comparison of Metabolic Markers Between Diabetic and Non-Diabetic Men

Significant differences were observed for several metabolic measures. Men with diabetes had higher body mass index, fasting glucose, systolic blood pressure, and diastolic blood pressure. The t test values were as follows: body mass index (t = 4.49, $p < 0.001$), fasting glucose (t = 2.70, $p < 0.01$), systolic blood pressure (t = 3.81, $p < 0.001$), and diastolic blood pressure (t = 5.88, $p < 0.001$). Total cholesterol, HDL cholesterol, and LDL cholesterol were significantly higher among participants with diabetes. However, triglycerides and VLDL cholesterol did not differ significantly between groups. These findings are summarised in Table 3.

Multivariable linear regression analysis was performed to assess whether diabetes status was independently associated with PSA after adjustment for age, body mass index, blood pressure, lipid fractions, and log-transformed fasting glucose. In the adjusted model for total PSA, diabetes status was not significantly associated with total PSA ($\beta = 0.09$, 95% CI -0.14-0.32, $p = 0.442$). Age remained a significant positive predictor of total PSA ($\beta = 0.03$, 95% CI 0.01–0.04, $p = 0.001$), while body mass index showed a significant inverse association (β = -0.04, 95% CI -0.07–0.01, $p = 0.009$). In the adjusted model for free PSA, diabetes status remained positively associated with free PSA, although the effect size was modest ($\beta = 0.11$, 95% CI 0.01–0.21, $p = 0.032$). Age was also positively associated with free PSA ($\beta = 0.01$, 95% CI 0.00-0.02, $p = 0.041$). No other cardio-metabolic variables were independently associated with PSA outcomes in the adjusted models. The regression results are presented in Table 4.

The multivariable regression model for total PSA demonstrated good overall fit (adjusted $R^2 = 0.19$; $F(9,136) = 4.78$, $p < 0.001$). Similarly, the model for free PSA showed a statistically significant overall fit, although with a lower explained variance (adjusted $R^2 = 0.13$; $F(9,136) = 3.11$, $p = 0.002$).

Table 2 Comparison of prostate-specific antigen levels between diabetic and non-diabetic men

Parameter	Diabetic ($n = 86$) Mean ± SD	Control ($n = 60$) Mean ± SD	t-value	*p*-value
Total PSA (ng/mL)	1.23 ± 1.03	0.94 ± 1.04	1.65	0.10
Free PSA (ng/mL)	0.39 ± 0.33	0.27 ± 0.36	2.19	0.03

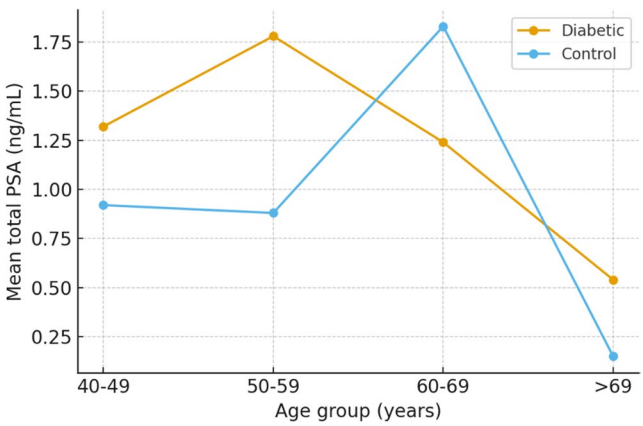

Fig. 1 Mean total PSA by age group (diabetic versus control)

Table 3 Comparison of cardio-metabolic markers between diabetic and non-diabetic men

Parameter	Diabetic ($n = 86$) Mean ± SD	Control ($n = 60$) Mean ± SD	t-value	*p*-value
BMI (kg/m²)	30.92 ± 4.62	27.84 ± 3.12	4.49	< 0.001
Fasting glucose (mmol/L)	13.08 ± 22.08	5.36 ± 0.61	2.70	< 0.01
Systolic BP (mmHg)	135.72 ± 14.89	127.40 ± 11.45	3.81	< 0.001
Diastolic BP (mmHg)	89.95 ± 13.31	78.83 ± 7.33	5.88	< 0.001
Total cholesterol (mmol/L)	4.83 ± 1.45	4.41 ± 1.08	2.26	0.03
HDL cholesterol (mmol/L)	1.30 ± 0.24	1.13 ± 0.27	3.93	< 0.001
LDL cholesterol (mmol/L)	3.03 ± 0.91	2.64 ± 0.75	2.72	< 0.01
Triglycerides (mmol/L)	1.07 ± 0.21	1.01 ± 0.27	1.44	0.15
VLDL (mmol/L)	0.48 ± 0.09	0.46 ± 0.12	1.37	0.17

Table 4 Multivariable linear regression analyses for total PSA and free PSA

Predictor	Total PSA, β (95% CI)	*p*-value	Free PSA, β (95% CI)	*p*-value
Diabetes status (diabetic vs. control)	0.09 (-0.14 to 0.32)	0.442	0.11 (0.01 to 0.21)	0.032
Age (years)	0.03 (0.01 to 0.04)	0.001	0.01 (0.00 to 0.02)	0.041
BMI (kg/m²)	-0.04 (-0.07 to -0.01)	0.009	-0.01 (-0.02 to 0.00)	0.058
Systolic BP (mmHg)	0.01 (0.00 to 0.02)	0.084	0.00 (-0.01 to 0.01)	0.611
Diastolic BP (mmHg)	-0.01 (-0.02 to 0.01)	0.392	0.00 (-0.01 to 0.01)	0.743
Total cholesterol (mmol/L)	0.06 (-0.04 to 0.16)	0.233	0.02 (-0.02 to 0.06)	0.317
HDL cholesterol (mmol/L)	0.12 (-0.18 to 0.42)	0.428	0.05 (-0.07 to 0.17)	0.402
LDL cholesterol (mmol/L)	0.05 (-0.07 to 0.17)	0.398	0.01 (-0.04 to 0.06)	0.711
Log fasting glucose	-0.08 (-0.25 to 0.09)	0.357	0.03 (-0.04 to 0.10)	0.386

Table 5 Age-stratified PSA, blood pressure, and fasting glucose values in diabetic men

Age group (years)	Total PSA (ng/mL) Mean ± SD	Systolic BP (mmHg) Mean ± SD	Diastolic BP (mmHg) Mean ± SD	Fasting glucose (mmol/L) Mean ± SD
40–49	1.32 ± 1.01	135.87 ± 7.45	95.00 ± 4.78	10.96 ± 5.00
50–59	1.78 ± 1.06	136.83 ± 20.93	98.33 ± 15.57	9.63 ± 4.19
60–69	1.24 ± 1.08	135.33 ± 15.95	87.50 ± 11.51	10.02 ± 5.28
> 69	0.54 ± 0.30	137.00 ± 10.92	85.50 ± 19.93	11.20 ± 5.14

Note: Total PSA: F (3,82) = 3.18, $p < 0.05$; diastolic BP: F (3,82) = 3.48, $p < 0.05$; log fasting glucose: F (3,82) = 0.32, $p > 0.05$

Age Group Differences in PSA, Blood Pressure, and Fasting Glucose

One-way ANOVA was used to examine age-related variation among diabetic participants. Significant differences were observed for total PSA and diastolic blood pressure (both $p < 0.05$). Post hoc comparisons showed that total PSA was lower in men older than 69 years compared with those aged 50–59 years. Diastolic blood pressure was also higher in the 50–59 year group than in the 60–69 and older than 69 year groups. Because fasting glucose was positively skewed, log-transformed values were used for inferential testing. The overall pattern of higher glucose levels among diabetic participants remained unchanged after transformation. These results are presented in Table 5. Figure 2 illustrates fasting glucose patterns across age groups in the diabetic population. Among non-diabetic controls, age-related variation was observed for systolic and diastolic blood pressure. Post hoc comparisons indicated that participants aged 60–69 years differed significantly from those aged 40–49 years for both measures.

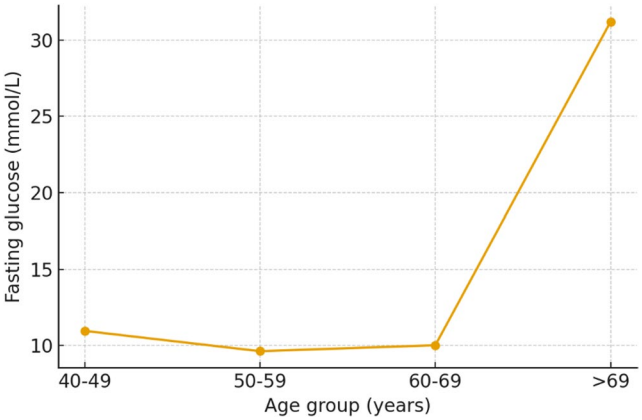

Fig. 2 Fasting glucose by age group (diabetic participants)

Associations Between Duration of Diabetes and PSA or Fasting Glucose

Spearman correlation coefficients showed no statistically significant relationship between duration of diabetes and total PSA ($r = 0.071$; $p = 0.52$). Scatterplots illustrating these relationships are presented in Fig. 3.

Associations Between BMI Categories, Lipid Profile, and Blood Pressure

BMI categories were not significantly associated with PSA classification. However, significant associations were observed between BMI categories and several lipid profile measures. Higher BMI categories were associated with higher total cholesterol, triglycerides, LDL cholesterol, and VLDL cholesterol, and with lower HDL cholesterol. The chi-square statistics are reported in Table 6. Figure 4 illustrates lipid profile distribution across BMI categories.

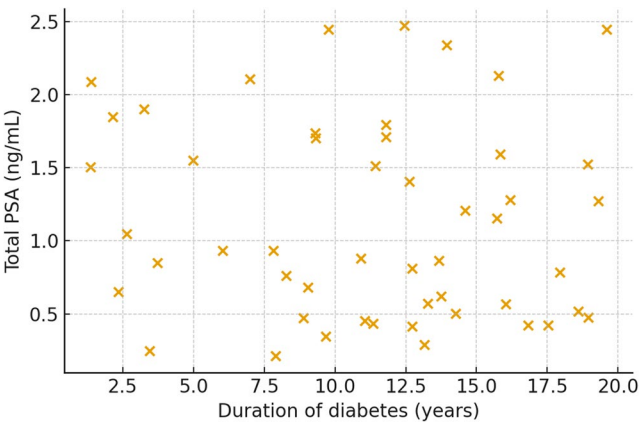

Fig. 3 Duration of diabetes and total PSA among diabetic participants

Table 6 Associations between BMI category and lipid parameters

Lipid measure	χ^2 value	*p*-value	Interpretation
Total cholesterol	23.99	<0.01	Significant association
Triglycerides	17.44	<0.01	Significant association
LDL cholesterol	17.44	<0.01	Significant association
HDL cholesterol	16.77	<0.05	Significant association
VLDL	22.47	<0.01	Significant association

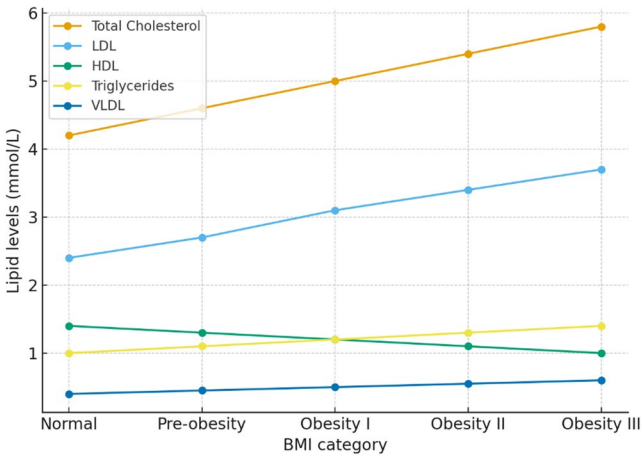

Fig. 4 Lipid profile distribution across BMI categories. Note: *TC = Total cholesterol; TG = Triglycerides; LDL = Low density lipoprotein; HDL = High density lipoprotein; VLDL = Very low-density lipoprotein*

Distribution of BMI Categories Across Age Groups in Diabetic Participants

BMI distribution varied across age groups among diabetic participants (Table 7). Younger men with diabetes were more likely to fall within the pre-obesity and obesity class I categories, while men aged 60 years and above were more widely distributed across higher obesity classes. A chi-square test indicated that age group was significantly associated with BMI category among diabetic men. This pattern suggests that age may influence obesity status within this population and may also contribute indirectly to metabolic variability.

Table 7 Association between BMI category and age group among diabetic participants ($n=86$)

Age group (years)	Normal, *n* (%)	Pre-obesity, *n* (%)	Obesity class I, *n* (%)	Obesity class II, *n* (%)	Obesity class III, *n* (%)
40–49	0 (0.0)	14 (16.3)	0 (0.0)	0 (0.0)	0 (0.0)
50–59	2 (2.3)	2 (2.3)	8 (9.3)	0 (0.0)	0 (0.0)
60–69	6 (7.0)	6 (7.0)	20 (23.3)	12 (14.0)	4 (4.7)
70–79	2 (2.3)	6 (7.0)	2 (2.3)	2 (2.3)	0 (0.0)

Note: $\chi^2 = 49.50$, $p<0.001$; Values are presented as frequency (percentage). Percentages are based on the total number of diabetic participants ($n=86$). BMI categories were defined according to World Obesity Federation criteria. A chi-square test indicated a statistically significant association between age group and BMI category.

Discussion

The findings of this study suggest that PSA behaviour in men with and without type 2 diabetes is shaped by a combination of age, adiposity, and broader cardio-metabolic factors. However, interpretation of the between-group findings must be made cautiously because of the substantial age imbalance between diabetic and non-diabetic participants. The adjusted analyses showed that diabetes status was not independently associated with total PSA, whereas age remained the strongest predictor. Diabetes status retained a modest positive association with free PSA after adjustment, although the magnitude of this effect was small. This finding reinforces the need to account for age when examining PSA in populations with heterogeneous demographic profiles. Previous studies reporting lower PSA levels in men with diabetes have often involved more closely age-matched samples, suggesting that observed differences may be sensitive to underlying population structure [26–28].

The study also showed that diabetic men had higher BMI, fasting glucose, and blood pressure than controls, which is consistent with the well-established clustering of cardio-metabolic risk factors in type 2 diabetes [29, 30]. The higher total cholesterol and LDL cholesterol levels among diabetic participants also align with the broader literature on diabetic dyslipidaemia [31, 33]. Although HDL cholesterol was also higher in the diabetic group, this pattern has not been consistently reported in previous studies and may reflect variation in treatment patterns, dietary habits, or body composition within this population [8, 30, 32, 33].

Age-related variation in PSA and selected cardio-metabolic markers was observed among diabetic participants. In particular, older age groups showed lower total PSA, while diastolic blood pressure also varied significantly across age categories. These findings differ from some studies in European and North American populations that report a more consistent rise in PSA with age and may reflect differences in adiposity, endocrine status, clinical characteristics, or other unmeasured factors in this setting [11–13, 23, 33].

No significant association was observed between duration of diabetes and PSA or fasting glucose. This is consistent with studies suggesting that the relationship between diabetes duration and prostate-related biomarkers is weak or inconsistent [34–36]. It is possible that treatment history, glycaemic control, or obesity exert greater influence than disease duration alone.

The significant association between BMI category and lipid abnormalities further supports the central role of adiposity in shaping cardio-metabolic risk in men with diabetes. This finding is relevant to PSA interpretation because obesity has been linked to lower measured PSA concentrations through haemodilution and hormonal mechanisms [37–43]. Overall, the results suggest that PSA should not be interpreted in isolation from the broader metabolic profile.

Although medication-related exclusions were applied, it was not always possible to verify complete treatment histories, particularly for statin or antihypertensive use initiated outside the hospital system. Some degree of misclassification is therefore possible. In addition, several factors known to influence PSA levels could not be fully controlled in this study. These include recent sexual activity, prostate manipulation, and physical activity such as cycling, all of which may transiently affect PSA concentrations. Undiagnosed benign prostatic hyperplasia also cannot be excluded. While these factors are unlikely to have differed systematically between groups, they may have contributed to measurement variability.

Limitations

The study has some important limitations. The most significant is the age imbalance between diabetic and control groups, which introduces residual confounding despite statistical adjustment. Because PSA and cardio-metabolic markers are strongly age-dependent, the lack of age matching limits the strength of causal inference and reduces confidence in unadjusted between-group comparisons. The use of consecutive sampling in a clinical setting also reduced control over group comparability.

The control group was smaller than initially planned, reflecting recruitment constraints during the study period. This may have reduced statistical power and contributed further to group imbalance. In addition, several pre-analytical factors that may influence PSA, including recent ejaculation, prostate manipulation, and physical exertion, were not standardised before sample collection. Undiagnosed benign prostatic conditions also could not be fully excluded. Finally, the case-control design and single time-point measurements prevent conclusions about temporal or causal relationships between metabolic factors and PSA.

Conclusion

These findings suggest that prostate-specific antigen levels in Nigerian men are influenced more strongly by age and adiposity than by diabetes status alone. Although free PSA was higher among diabetic men in unadjusted analyses, this finding should be interpreted cautiously in view of the age imbalance and residual confounding. Clinical interpretation of PSA in populations with a high burden of metabolic disease should therefore consider broader cardio-metabolic context rather than diabetes status alone. Future studies should use age-matched designs, larger samples, and longitudinal approaches to clarify these relationships.

Author Contributions OCU and UAE contributed to the conception of the study; OCU wrote the initial draft of the manuscript, led the data analysis, recruited study participants and data collection; CNO performed the statistical analysis; PA and NCU provided critical resources and materials for the study; ENO contributed to the literature review, helped with writing, and participated in the supervision of the study; CUO assisted with the data collection; OJC, JO and UAE assisted in the statistical analysis review and editing; CVU and OAO contributed to selecting study participants and data collection; CO contributed to the ethical review application and committee approval, selecting study participants, and data collection; UAE and JO contributed to the literature review, helped with writing, and participated in the supervision of the study. All authors reviewed the draft manuscript and provided constructive discussions. All authors have read and approved the manuscript for publication.

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Data Availability The original contributions presented in the study are included in the article/supplementary material, further inquiries can be directed to the corresponding or first author.

Code Availability Not applicable.

Declarations

Ethics approval The study was approved by the College of Medicine Research and Ethics Committee, University of Nigeria Enugu Campus (Reference: 073/07/2019).

Consent to participate All study participants provided written informed consent to take part in the research.

Written consent for publication All study participants gave consent for the data collected to be used in publication in anonymized form.

Competing interests The authors declare no competing interests.

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