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Diagnostic Performance of a Combined KLK2, miR-21, PSA, CRP, Albumin, AGR, and GGT Biomarker Panel for Distinguishing Prostate Cancer from Benign Prostatic Hyperplasia in Nigerian Men

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Keywords: prostate cancer, benign prostatic hyperplasia, biomarker panel, Kallikrein-related peptidase 2, diagnostic accuracy, ROC curve, Nigeria

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Study design: Cross-sectional, comparative case-control study.

Article type: Research (Original Article)

Abstract

Background: Prostate-specific antigen (PSA) has limited specificity for distinguishing prostate cancer from benign prostatic hyperplasia (BPH) in African populations, where high baseline PSA and a high background prevalence of BPH inflate false-positive results. Whether a combined panel of PSA, Kallikrein-related peptidase 2 (KLK2), miR-21, C-reactive protein (CRP), albumin, the albumin-to-globulin ratio (AGR), and gamma-glutamyl transferase (GGT) improves on PSA alone for distinguishing malignant from non-malignant prostatic disease in Nigerian men has not been established.

Methods: In this cross-sectional case-control study, 90 men (30 histologically confirmed prostate cancer cases, 30 BPH cases, and 30 healthy controls) were recruited from four hospitals in Abuja, Nigeria. Serum PSA, KLK2, CRP, albumin, and GGT were measured by immunoassay, circulating miR-21 by quantitative PCR, and AGR calculated from albumin and globulin. Receiver operating characteristic (ROC) curve analysis established the diagnostic accuracy of each biomarker and of five incremental combined models against a non-malignant reference group (BPH and healthy controls combined), with the DeLong test used to compare correlated areas under the curve (AUC). Firth's penalized logistic regression identified independent predictors of cancer status, and five-fold cross-validation assessed model stability.

Results: PSA, KLK2, and CRP each differed significantly across every pairwise group comparison, including BPH versus cancer (all $p<0.01$). PSA alone achieved the highest diagnostic accuracy (AUC 0.990; sensitivity 93.3%, specificity 100.0%), exceeding KLK2 (AUC 0.913), CRP (AUC 0.868), GGT (AUC 0.721), miR-21 (AUC 0.688), and AGR (AUC 0.502). Each of PSA, KLK2, miR-21, CRP, and GGT was independently associated with cancer status on univariate analysis (all $p<0.05$), but none retained significance after mutual adjustment in a multivariable model, reflecting strong intercorrelation among PSA, KLK2, and CRP (Spearman rho 0.78-0.81). Combining KLK2, miR-21, CRP, and the biochemical markers with PSA did not improve diagnostic accuracy over PSA alone, and the full combined panel achieved a significantly lower AUC than PSA alone (DeLong test, $p=0.029$). A reduced five-variable model (PSA, KLK2, miR-21, CRP, age) nonetheless achieved strong internal discrimination (AUC 0.901) and was translated into a nomogram.

Conclusions: In this late-presenting Nigerian cohort, PSA alone achieved near-ceiling diagnostic accuracy for distinguishing prostate cancer from non-malignant prostatic disease, and combining it with KLK2, miR-21, CRP, and biochemical markers did not improve, and numerically reduced, accuracy. KLK2 and CRP nonetheless carry genuine, independently significant diagnostic information and distinguished cancer from BPH on direct pairwise comparison, and a reduced composite model built entirely on locally available assays may hold feasibility-stage value for earlier-presenting or grey-zone patients, pending external validation.

Keywords: prostate cancer; benign prostatic hyperplasia; biomarker panel; Kallikrein-related peptidase 2; diagnostic accuracy; ROC curve; Nigeria

Background

Prostate-specific antigen (PSA) remains the mainstay of prostate cancer screening worldwide, but its specificity for malignancy is limited, particularly in the diagnostic grey zone of 4-10 ng/mL, where only 25-40% of elevated results reflect true cancer rather than benign prostatic hyperplasia (BPH) or other confounding processes [1]. This limitation is especially consequential in African populations: men of African ancestry carry higher baseline PSA than Western counterparts even when apparently healthy, and the high background prevalence of BPH among Nigerian men over 60 years (40-60%) further inflates PSA values, contributing to unnecessary biopsy in an estimated 60-70% of cases with elevated PSA [2, 3].

These limitations have prompted interest in complementary biomarkers capable of improving specificity without sacrificing the sensitivity that makes PSA clinically useful. Kallikrein-related peptidase 2 (KLK2) is a serine protease approximately 80% homologous to PSA but more specific to malignant prostatic epithelium, and forms a core component of the validated 4Kscore panel, which achieves an area under the receiver operating characteristic curve (AUC) of 0.82 for high-grade disease in European cohorts and reduces unnecessary biopsy by 30-43% [4-6]. MicroRNA-21 (miR-21), a small non-coding RNA that silences the tumour suppressors PTEN, PDCD4, and TPM1, shows reported sensitivity of 81-92% and specificity of 78-88% as a serum-based marker in other populations [7, 8]. C-reactive protein (CRP), an acute-phase marker of the systemic inflammation that accompanies tumour progression, and gamma-glutamyl transferase (GGT), a hepatobiliary enzyme linked to oxidative stress and more aggressive disease, add complementary information on the inflammatory and metabolic correlates of malignancy [9, 10]. Protein-fraction markers, including albumin and the albumin-to-globulin ratio (AGR), have shown prognostic value in other cancers, though their evidence base in prostate cancer specifically is more limited [11].

Multi-marker panels incorporating these analytes, most notably the 4Kscore and the Stockholm3 model, have demonstrated meaningful incremental value over PSA alone in cohorts enriched for grey-zone PSA values, where PSA's discriminatory power is genuinely limited [5, 6, 12]. Whether this incremental value generalizes to a Nigerian clinical population, where diagnostic delay is common and presenting PSA is often far outside the grey zone, has not been established. To our knowledge, no Nigerian study has evaluated a combined panel of KLK2, miR-21, CRP, albumin, AGR, and GGT alongside PSA for distinguishing prostate cancer from BPH, the clinically relevant differential diagnosis that drives the large majority of PSA-triggered biopsies in this setting [13].

This study therefore evaluated the individual and combined diagnostic performance of PSA, KLK2, miR-21, CRP, albumin, AGR, and GGT for distinguishing prostate cancer from non-malignant prostatic disease among men attending four hospitals in Abuja, Nigeria, with particular attention to the pairwise comparison between confirmed prostate cancer and BPH, and assessed the feasibility of a reduced composite risk model built on assays already available within existing hospital laboratory infrastructure.

Methods

Study design, setting and participants

This was a cross-sectional, comparative case-control study conducted across four tertiary and secondary hospitals in the Federal Capital Territory (FCT) of Nigeria: the University of Abuja Teaching Hospital (UATH), Gwagwalada; National Hospital Abuja; Garki Hospital, Abuja; and General Hospital, Bwari. Sample collection ran for twelve months, from February 2025 to February 2026. Ethical clearance was obtained from the Health Research Ethics Committee of UATH (UATH/HREC/PR/583) and the FCT Health Research Ethics Committee

(FHREC/2025/01/64/13-03-25), and written informed consent was obtained from every participant.

Adult Nigerian men aged 40 years and older were recruited into three groups: prostate cancer cases, with disease confirmed by histopathology and graded using the Gleason scoring system, and no prior definitive treatment at enrolment; BPH cases, confirmed clinically and ultrasonographically, with PSA below 10 ng/mL, no biopsy suspicion of malignancy, and age-matched to within ±5 years of the cancer group; and healthy controls, with PSA below 2.5 ng/mL, a normal digital rectal examination, and no urological symptoms. Exclusion criteria, applied uniformly across all three groups, included acute febrile illness or recent hospitalization, known liver or renal disease unrelated to the disease under study, any malignancy other than prostate cancer, and current use of medications known to affect the study biomarkers. Consecutive eligible patients were enrolled until each group's target of 30 participants was reached, consistent with a minimum sample size of 32 per group indicated by a priori power calculation (α=0.05, power=80%). The final analytic sample comprised 90 men: 30 healthy controls, 30 BPH patients, and 30 histologically confirmed prostate cancer patients.

Biological sampling and biomarker assays

Venous blood (10 mL) was drawn from each participant by aseptic technique: 4 mL into a K3-EDTA tube for plasma and buffy-coat separation, and 6 mL into a plain serum-separator tube. Serum aliquots were stored at -80°C until analysis. Serum PSA, KLK2, and CRP were quantified using validated sandwich enzyme-linked immunosorbent assay (ELISA) kits, with absorbance read spectrophotometrically at 450 nm. Albumin, total protein, and GGT were measured by standard colorimetric and kinetic assays on an automated clinical chemistry analyser, and globulin and AGR were derived by calculation. Circulating miR-21 expression was quantified by quantitative real-time PCR (qPCR) from plasma-extracted RNA and is

reported as cycle threshold (Ct) value, such that a lower Ct value corresponds to higher miR-21 expression.

Statistical analysis

Data were analyzed using IBM SPSS Statistics (version 27.0) and, for ROC curve comparison, MedCalc software. The normality of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed variables were compared using one-way analysis of variance (ANOVA), and non-normally distributed variables using the Kruskal-Wallis test with Dunn's post-hoc test (Bonferroni-corrected) for pairwise comparisons, including the BPH-versus-cancer comparison specifically. The diagnostic performance of each biomarker, and of five incremental combined models, was evaluated against a non-malignant reference group combining BPH and healthy control participants, using ROC curve analysis; the area under the curve (AUC) and its 95% confidence interval were calculated for each marker and model, and optimal cut-offs were determined using the Youden index (J = sensitivity + specificity – 1). The AUCs of combined models were compared with PSA alone using the DeLong test for correlated ROC curves [14]. Independent associations between biomarker levels and cancer status were assessed by univariate and multivariable binary logistic regression, with biomarker levels and age entered as predictors and results expressed as odds ratios (OR) with 95% confidence intervals; where standard maximum-likelihood estimation failed to converge because of quasi-complete separation between outcome groups, Firth's penalized logistic regression was applied as a pre-specified contingency method [15, 16]. Internal validation of the combined-panel model was performed using five-fold cross-validation. A p-value below 0.05 was considered statistically significant throughout.

Results

Biomarker levels and pairwise group comparisons, including BPH versus cancer

PSA, KLK2, CRP, and GGT each rose progressively from healthy controls through BPH to prostate cancer patients (all $p<0.001$), and miR-21 Ct values fell progressively across the same groups, consistent with rising miR-21 expression as disease advanced. On direct pairwise comparison, the differential diagnosis of central interest to this study, PSA, KLK2, and CRP each differed significantly between BPH and cancer patients specifically, while miR-21 and GGT did not distinguish BPH from cancer on pairwise testing despite differing significantly from healthy controls (Table 1).

Table 1 Pairwise comparison of biomarker levels between study groups, including BPH versus prostate cancer

Biomarker	Healthy vs BPH	Healthy vs Cancer	BPH vs Cancer
PSA	<0.0001*	<0.0001*	0.0001*
KLK2	<0.0001*	<0.0001*	0.0052*
miR-21	<0.0001*	<0.0001*	1.000
CRP	<0.0001*	<0.0001*	0.0296*
GGT	0.0147*	<0.0001*	0.3657

*Values are Bonferroni-corrected p-values from Dunn's post-hoc test following a significant Kruskal-Wallis test. *p<0.05.*

Diagnostic performance of individual biomarkers

Table 2 presents the diagnostic performance of each biomarker for distinguishing prostate cancer from the combined non-malignant reference group (BPH and healthy controls). PSA achieved the highest accuracy (AUC 0.990; sensitivity 93.3%, specificity 100.0%), followed by KLK2 (AUC 0.913) and CRP (AUC 0.868). GGT and miR-21 showed moderate discrimination (AUC 0.721 and 0.688, respectively), and AGR performed no better than chance (AUC 0.502).

Table 2 Diagnostic performance of individual biomarkers for distinguishing prostate cancer from non-malignant prostatic disease (BPH and healthy controls combined)

Marker	Cut-off	Sensitivity	Specificity	PPV	NPV	AUC	95% CI
PSA (ng/mL)	13.20	93.3%	100.0%	100.0%	96.8%	0.990	0.971-1.000
KLK2 (ng/mL)	1.30	70.0%	95.0%	87.5%	86.4%	0.913	0.844-0.965
miR-21 (Ct)	32.42	40.0%	100.0%	100.0%	76.9%	0.688	0.553-0.801
CRP (mg/L)	1.80	70.0%	98.3%	95.5%	86.8%	0.868	0.768-0.945
GGT (IU/L)	41.10	56.7%	81.7%	60.7%	79.0%	0.721	0.596-0.838
AGR	5.77	10.0%	100.0%	100.0%	69.0%	0.502	0.373-0.630

PPV, positive predictive value; NPV, negative predictive value; AUC, area under the receiver operating characteristic curve; CI, confidence interval. Reference group: BPH and healthy controls combined (n=60) versus prostate cancer (n=30).

Independent associations with cancer status

On univariate Firth logistic regression (Table 3), PSA, KLK2, miR-21, CRP, and GGT were each significantly associated with cancer status; age and AGR were not. When all seven predictors were entered simultaneously in a multivariable model (Table 4), none retained independent statistical significance, despite each being significant in isolation; every 95% confidence interval for the adjusted odds ratios crossed the line of no effect. This pattern is consistent with strong mutual intercorrelation among PSA, KLK2, and CRP (Spearman rho 0.78-0.81 across the full cohort), a textbook signature of collinear predictors competing for shared explained variance rather than evidence against the underlying biological association of these markers with cancer status.

Table 3 Univariate association between biomarker levels and prostate cancer status (Firth's penalized logistic regression)

Variable	OR	95% CI	p-value
Age (years)	1.026	0.983-1.071	0.246
PSA (ng/mL)	1.093	1.042-1.148	0.0003*
KLK2 (ng/mL)	1.545	1.076-2.217	0.018*
miR-21 (per unit ΔCt)	0.499	0.312-0.798	0.004*

CRP (mg/L)	3.911	2.005-7.630	<0.001*
GGT (IU/L)	1.022	1.003-1.040	0.021*
AGR	1.071	0.699-1.641	0.752

**p<0.05. OR, odds ratio; CI, confidence interval.*

Table 4 Multivariable association between biomarker levels and prostate cancer status, all seven predictors mutually adjusted (Firth's penalized logistic regression)

Predictor	Adjusted OR	95% CI	p-value
PSA (ng/mL)	1.008	0.990-1.027	0.378
KLK2 (ng/mL)	0.577	0.232-1.435	0.237
miR-21 (per unit ΔCt)	0.700	0.398-1.230	0.215
CRP (mg/L)	1.873	0.735-4.773	0.188
GGT (IU/L)	1.003	0.992-1.015	0.560
AGR	0.921	0.561-1.511	0.744
Age (years)	1.024	0.976-1.075	0.329

Diagnostic accuracy of combined biomarker panels

Table 5 compares five incremental biomarker combinations. PSA alone achieved the highest AUC (0.990); progressively adding KLK2, miR-21, CRP, and the remaining biochemical markers did not improve, and in each case numerically reduced, apparent discrimination, a pattern confirmed by independent five-fold cross-validation. The DeLong test (Table 6) confirmed that PSA alone significantly outperformed KLK2 alone, the PSA+KLK2 combined model, and the full combined biomarker panel.

Table 5 Comparison of five incremental biomarker combinations as prediction models for prostate cancer versus non-malignant prostatic disease

Model	Variables	In-sample AUC	5-fold CV AUC (mean±SD)	Specificity
1	PSA	0.990	0.992±0.017	100.0%
2	PSA + KLK2	0.926	0.967±0.027	100.0%
3	PSA + KLK2 + miR-21	0.866	0.944±0.033	100.0%
4	PSA + KLK2 + miR-21 + CRP	0.880	0.914±0.054	100.0%

5	Full panel (+ GGT, AGR)	0.900	0.964±0.046	100.0%

AUC, area under the curve; CV, cross-validation; SD, standard deviation.

Table 6 Comparison of receiver operating characteristic curves, PSA versus combined models (DeLong's test)

Comparison	ΔAUC	z	p-value
PSA vs KLK2	0.077	2.851	0.0044*
PSA vs PSA+KLK2	0.064	2.351	0.0187*
PSA vs Combined Panel (Model 5)	0.090	2.178	0.0294*

**p<0.05.*

Feasibility of a reduced composite risk model

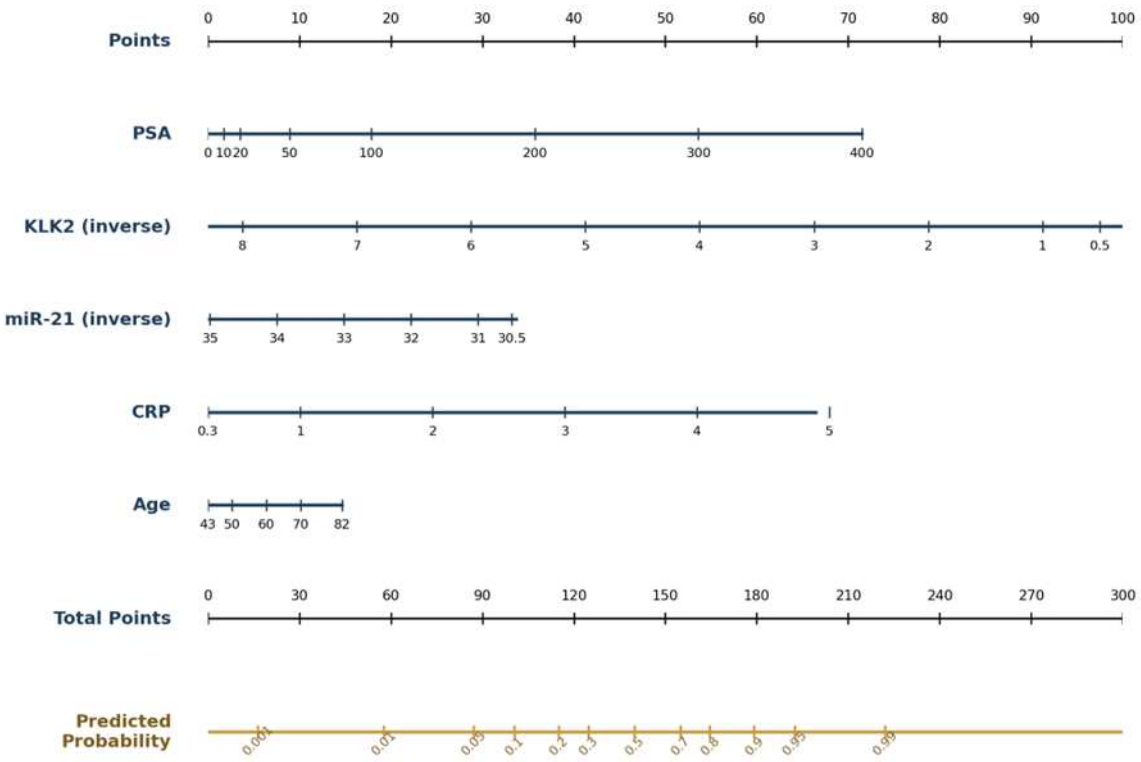
A reduced model retaining PSA, KLK2, miR-21, CRP, and age achieved strong overall discrimination (AUC 0.901) despite no individual coefficient reaching significance after mutual adjustment (Table 7), consistent with prediction-modelling methodology in which a composite score's value is judged by aggregate discrimination rather than individual coefficient significance [17]. This model was translated into a nomogram (Figure 1) permitting a total point score, and corresponding predicted probability of cancer, to be derived directly from a patient's PSA, KLK2, miR-21, CRP, and age using assay platforms already available within the four participating hospitals.

Table 7 Final reduced risk-prediction model (PSA, KLK2, miR-21, CRP, age)

Predictor	β (coefficient)	SE	OR	p-value
Intercept	11.192	9.820	72515.99	0.254
PSA (ng/mL)	0.010	0.010	1.010	0.283
KLK2 (ng/mL)	-0.699	0.492	0.497	0.156
miR-21 (per unit ΔCt)	-0.410	0.289	0.664	0.156
CRP (mg/L)	0.808	0.497	2.243	0.104
Age (years)	0.021	0.025	1.021	0.407

SE, standard error; OR, odds ratio. KLK2 and miR-21 coefficients are negative in this multivariable model as a product of collinearity with PSA and CRP rather than a genuine protective effect.

Figure 1 Nomogram for predicted probability of prostate cancer,



Nomogram for predicted probability of prostate cancer, derived from Table 7. KLK2 and miR-21 are labelled "(inverse)" because their model coefficients were negative, reflecting collinearity with PSA and CRP rather than a genuine protective effect.

Discussion

This study evaluated the individual and combined diagnostic performance of a seven-marker panel, PSA, KLK2, miR-21, CRP, albumin, AGR, and GGT, for distinguishing prostate cancer from non-malignant prostatic disease among men attending four hospitals in Abuja, Nigeria, with particular attention to the pairwise distinction between confirmed cancer and BPH. Three findings stand out. First, PSA, KLK2, and CRP each distinguished cancer from BPH on direct pairwise comparison, not only from healthy controls, confirming that their diagnostic signal extends to the clinically decisive differential diagnosis. Second, PSA alone achieved near-ceiling diagnostic accuracy in this cohort, exceeding every other individual marker and every

combined panel tested. Third, combining KLK2, miR-21, CRP, and the biochemical markers with PSA did not improve, and in several configurations measurably reduced, diagnostic accuracy relative to PSA alone.

PSA's near-ceiling performance (AUC 0.990) should be read as a reflection of this study's case-mix rather than a genuine departure from the extensive literature documenting PSA's limited specificity in the 4-10 ng/mL grey zone [1, 18]. The great majority of confirmed cancer cases in this cohort presented with PSA values far outside that ambiguous range, consistent with the substantial diagnostic delay and markedly elevated presenting PSA repeatedly documented in Nigerian prostate cancer series [18, 19]. KLK2's individual performance (AUC 0.913) was numerically higher than its incremental contribution within the validated 4Kscore panel [5, 6], plausibly because its greater specificity for prostatic epithelium relative to PSA should manifest most clearly where malignant burden, and therefore KLK2 secretion, is already high [20]. CRP's performance (AUC 0.868) corroborated the inflammatory-oncogenesis literature linking systemic inflammation to prostate cancer progression [9, 21], while miR-21 and GGT showed only moderate discrimination and AGR performed no better than chance, consistent with reports that AGR's prognostic value concentrates in populations enriched for advanced or recurrent disease [11].

The apparent failure of combined panels to outperform PSA alone warrants careful interpretation. Univariate analysis confirmed that PSA, KLK2, CRP, GGT, and miR-21 were each independently associated with cancer status; the loss of significance for every predictor once all seven were entered simultaneously is a textbook signature of collinearity, not evidence that these markers lack biological relevance to malignant transformation. PSA, KLK2, and CRP were strongly and mutually intercorrelated (pairwise Spearman rho 0.78-0.81) across the full cohort, meaning their diagnostic information substantially overlaps in a population such as this one, where PSA already operates near ceiling performance. When a single marker already

achieves near-perfect discrimination, there is mathematically little room for additional correlated markers to add incremental value, however genuine their individual biological association with disease may be. This finding stands in productive tension with, rather than simple contradiction of, the international multi-marker literature: the 4Kscore's incremental gain over PSA alone was demonstrated specifically in cohorts enriched for grey-zone PSA values, where PSA genuinely struggles and KLK2 has room to add information PSA cannot supply [5, 22]. This study, dominated by advanced-stage, high-PSA presentations, sits close to the opposite case, and the combined-panel AUCs, each numerically lower than PSA alone and significantly so for the full panel on DeLong testing, should be read as evidence that combined-panel value in this population is conditional on case-mix, not as evidence against the biological relevance of KLK2, CRP, or miR-21.

Despite this, PSA, KLK2, and CRP each distinguished BPH from cancer directly, the specific differential diagnosis that drives the large majority of biopsy referrals in Nigerian urological practice, whereas miR-21 and GGT, though each significantly elevated relative to healthy controls, did not separate BPH from cancer on pairwise testing. This distinction matters clinically: a marker's ability to separate disease from health is a necessary but not sufficient basis for diagnostic utility, since the decision clinicians actually face when interpreting an elevated PSA is whether the patient has cancer or BPH, not whether the patient is diseased or healthy. KLK2 and CRP's demonstrated capacity to make this specific distinction, even where they did not improve on PSA's overall AUC, supports their candidacy as reflex or adjunct tests in grey-zone or ambiguous presentations, where PSA alone is least reliable, rather than as replacements for PSA across all presentations.

A reduced model retaining PSA, KLK2, miR-21, CRP, and age achieved strong aggregate discrimination (AUC 0.901) and was translated into a nomogram requiring no computational infrastructure beyond a printed chart, a deliberate methodological choice intended to close the

gap between statistical modelling and bedside usability in resource-constrained settings [17, 23]. Every assay contributing to this reduced model is already performed or readily performable using laboratory infrastructure at the four participating FCT hospitals [13], meaning that adoption would require staff training and quality-assurance protocols rather than new capital equipment. We present this reduced model and its nomogram as a feasibility-stage output, evidence that a composite score can be derived and shows reasonable internal discrimination in this sample, rather than as a validated clinical-utility claim; that would require external validation in an independent cohort, a prospective implementation study, and formal cost-effectiveness analysis, none of which this cross-sectional design can provide.

This study has its limitations. The reference group against which diagnostic accuracy was calculated combined BPH and healthy control participants rather than isolating BPH alone; while the pairwise comparisons in Table 1 confirm that PSA, KLK2, and CRP each distinguish cancer from BPH specifically, a dedicated ROC analysis restricted to a BPH-only reference group, ideally in a larger sample, would provide a more clinically direct estimate of diagnostic accuracy for this specific differential diagnosis. Recruitment relied on consecutive sampling of eligible patients at four purposively selected hospitals rather than population-based random sampling, a standard and defensible approach for hospital-based case-control studies but one that may limit generalizability beyond men who present for care at these specific facilities. The cross-sectional design captured biomarker levels at a single timepoint and cannot establish temporal or causal relationships. Finally, the cohort was drawn from a single geopolitical zone and was dominated by late-presenting, high-PSA disease; this shaped the diagnostic performance reported for PSA and KLK2 specifically and limits generalizability to earlier-presenting or grey-zone populations, where these markers' comparative and combined value is most clinically relevant and remains to be established prospectively.

Conclusions

Among men attending four hospitals in Abuja, Nigeria, PSA alone achieved near-ceiling diagnostic accuracy for distinguishing prostate cancer from non-malignant prostatic disease, and combining it with KLK2, miR-21, CRP, albumin, AGR, and GGT did not improve, and in several configurations reduced, diagnostic accuracy. KLK2 and CRP nonetheless carry genuine, independently significant diagnostic information and distinguished prostate cancer from BPH directly on pairwise comparison, supporting their candidacy as adjunct or reflex tests for grey-zone or ambiguous PSA presentations rather than as blanket replacements for PSA testing. A reduced composite model (PSA, KLK2, miR-21, CRP, age), built entirely on assays already available within existing FCT hospital infrastructure, achieved strong internal discrimination and was translated into a bedside-usable nomogram; this feasibility-stage tool merits external validation, ideally in a cohort enriched for grey-zone PSA presentations, before any claim of validated clinical utility can be made.

List of abbreviations

AGR: Albumin-to-globulin ratio; AUC: Area under the receiver operating characteristic curve; BPH: Benign prostatic hyperplasia; CI: Confidence interval; CRP: C-reactive protein; CV: Cross-validation; ELISA: Enzyme-linked immunosorbent assay; FCT: Federal Capital Territory; GGT: Gamma-glutamyl transferase; KLK2: Kallikrein-related peptidase 2; miR-21: microRNA-21; NPV: Negative predictive value; OR: Odds ratio; PPV: Positive predictive value; PSA: Prostate-specific antigen; qPCR: Quantitative polymerase chain reaction; ROC: Receiver operating characteristic; SD: Standard deviation; SE: Standard error.

Declarations

Ethics approval and consent to participate

Ethical clearance was obtained from the Health Research Ethics Committee of the University of Abuja Teaching Hospital (UATH/HREC/PR/583) and the Federal Capital Territory Health

Research Ethics Committee (FHREC/2025/01/64/13-03-25). Written informed consent to participate was obtained from all participants.

Consent for publication

Not applicable. This manuscript does not contain any individual person's data in any identifiable form.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

SAA conceptualized the study, conducted the investigation and formal analysis, and drafted the manuscript. MFO supervised the study and contributed to conceptualization, methodology, and manuscript review. OBO, MUB, INA, SAA(2), BIS, MAM and DPO contributed to investigation, data curation, and manuscript review. All authors read and approved the final manuscript.

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Authors' information

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