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Prevalence and Factors Associated With Incidental Prostate Cancer After Benign Prostatic Hyperplasia Surgery: A Five-Year Retrospective Study in Addis Ababa, Ethiopia

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Abstract

Background

Incidental prostate cancer (IPC), defined as cancer unexpectedly identified on histopathology after surgery for presumed benign prostatic hyperplasia (BPH), has been widely studied internationally, but data from sub-Saharan Africa remain scarce, and none exist from Ethiopia. This study examined the prevalence, predictors, clinicopathological features, and management of IPC among patients undergoing transurethral resection of the prostate (TURP) or transvesical prostatectomy (TVP) for presumed BPH.

Methods

This retrospective cross-sectional study included 239 patients who underwent TURP or TVP for presumed BPH at MCM Comprehensive Specialized Hospital, Addis Ababa, Ethiopia, between January 2021 and December 2025. Sociodemographic, clinical, laboratory, and procedural data were abstracted from medical records. Bivariable comparisons used chi-square or Fisher's exact tests and t-tests or Mann–Whitney U tests, as appropriate; univariable and multivariable logistic regression estimated crude and adjusted odds ratios (AORs) with 95% confidence intervals (CIs). Model fit and discrimination were assessed, and sensitivity analyses examined robustness to prostate-specific antigen (PSA) transformation and influential observations

Results

IPC was identified in 23 of 239 patients (9.6%), with no significant difference by procedure (TURP 11.1% vs. TVP 8.2%, $p = 0.445$). Patients with IPC had significantly higher rates of hypertension (47.8% vs. 26.4%, $p = 0.030$), hydronephrosis (39.1% vs. 14.4%, $p = 0.006$), acute kidney injury (39.1% vs. 8.3%, $p < 0.001$), and higher PSA and PSA density (both $p < 0.001$). On multivariable analysis, preoperative PSA (AOR = 1.25 per ng/mL, 95% CI: 1.14–1.38), diabetes mellitus (AOR = 5.28, 95% CI: 1.64–16.95), and hydronephrosis (AOR = 3.92, 95% CI: 1.29–11.94) were independently associated with IPC; age was not (AOR = 1.19 per decade, $p = 0.459$). The model showed good discrimination (area under the curve [AUC] = 0.838) and calibration (Hosmer–Lemeshow $p = 0.162$), with findings robust across sensitivity analyses. Among IPC cases, 95.7% had International Society of Urological Pathology (ISUP) Grade Group ≥ 2, yet no patient received active surveillance, radical prostatectomy, radiotherapy, or documented staging investigation; management was uniformly based on androgen deprivation.

Conclusions

IPC affected nearly 1 in 10 men undergoing BPH surgery, with PSA, diabetes mellitus, and hydronephrosis independently associated with it; PSA density > 0.15 had 90% specificity but only 48%

sensitivity. Most cases were clinically significant, yet none received staging or definitive local therapy, highlighting the need for structured incidental-cancer care pathways in similar resource-limited settings.

Introduction

Prostate cancer is the second most common cancer among men worldwide and a leading cause of cancer death, ranking among the most frequently diagnosed male cancers in Ethiopia(1, 2). Benign prostatic hyperplasia (BPH), a near-universal condition in aging men, is treated surgically most often by transurethral resection of the prostate (TURP) or transvesical prostatectomy (TVP) when symptoms are refractory to medical therapy or complications arise (3, 4).

Because a definitive diagnosis of prostate cancer requires histopathological examination, cancer that was clinically unsuspected before surgery may be discovered incidentally in tissue removed during BPH surgery termed incidental prostate cancer (IPC) (5–7). Reported IPC prevalence varies widely across populations, and the factors that predict it, along with how it is subsequently managed, differ by setting (8).

Data on IPC from sub-Saharan Africa remain limited to a single comparable report, from Somalia (9); no data exist from Ethiopia. Given known regional differences in PSA screening practices, diagnostic capacity, and access to definitive oncologic treatment, locally generated evidence is needed to inform preoperative counseling, pathological sampling practices, and post-diagnosis care pathways in this setting.

This study aimed to determine the prevalence of IPC among patients undergoing TURP or TVP for presumed BPH at MCM Comprehensive Specialized Hospital, to identify factors independently associated with IPC, and to describe the clinicopathological characteristics and management of the incidentally diagnosed cases.

Methods

Study design, setting and aim

This study was an institution-based, retrospective, single-center, cross-sectional study conducted to assess the prevalence and determinants of IPC among men undergoing TURP or TVP for presumed BPH at MCM Comprehensive Specialized Hospital in Addis Ababa, Ethiopia. The study included 239 patients who underwent TURP or TVP between January 2021 and December 2025.

Participants

The study included male patients who underwent TURP or TVP for presumed BPH during the study period. Patients were eligible if they had available histopathological examination of the resected prostate tissue and met the predefined inclusion criteria. Patients with a known diagnosis of prostate cancer before surgery or incomplete medical or histopathological records were excluded. Operating-room

registry logbooks were screened for all patients who underwent TURP or TVP for presumed BPH between January 2021 and December 2025 (n = 423). Of these, 184 were excluded because of a known diagnosis of prostate cancer before surgery, missing histopathology report, or incomplete medical records. The remaining 239 patients, comprising all eligible patients during the study period, were included in the analysis.

Study variables and outcome assessment

The primary outcome of the study was the presence of IPC, defined as prostate cancer unexpectedly identified on histopathological examination of prostate tissue obtained during surgery performed for presumed BPH. IPC was categorized as a binary outcome (yes/no). Among patients diagnosed with IPC, histopathological characteristics like Gleason score, and International Society of Urological Pathology (ISUP) were additionally assessed descriptively. Gleason score was restratified based on risk.

Potential predictors of IPC included sociodemographic, clinical, laboratory, and procedural characteristics. These included age, body mass index (BMI), smoking history, preoperative PSA level, PSA density, prostate volume, digital rectal examination (DRE) findings, diabetes mellitus, hypertension, cardiac disease, urinary tract infection (UTI), hydronephrosis, acute kidney injury (AKI), and type of prostate surgery (TURP or TVP), as available in the medical records.

Data sources

All resected prostatic tissue (TURP chips or the TVP specimen) was sent for histopathological examination, and the reports were reviewed to determine the presence of IPC. Tumor grade was recorded as the ISUP Grade Group reported in the pathology report and categorized as low (Grade Group 1), intermediate (Grade Groups 2–3), or high risk (Grade Groups 4–5). Reports were issued by qualified/certified pathologist at the study institute.

Data Quality Control

- A structured and pretested data abstraction checklist was used for data collection.
- All study variables were clearly defined and standardized coding procedures were followed. Operational definitions specified in the study protocol were applied consistently during data abstraction.
- Data collectors and supervisors were oriented regarding the study objectives, eligibility criteria, variable definitions, and data abstraction procedures.
- Collected data were reviewed for completeness, consistency, and accuracy before analysis. Discrepancies and missing information identified during the review were checked against the original medical records and histopathology reports where available.

Sample size

No sample size calculation was performed. All patients who met the eligibility criteria during the study period were included (census sampling), yielding 239 patients, of whom 23 had IPC. Because of the limited number of IPC events, the multivariable model was restricted to four prespecified variables (events per variable = 5.75) to limit overfitting.

This calculation addressed precision of the prevalence estimate only. It was not designed to provide power for the regression analyses; because of the limited number of IPC events, the multivariable model was therefore restricted to four prespecified variables.

Data Analysis

Data were entered, cleaned, coded, and analyzed using IBM SPSS Statistics version 27 and Python version 3.12 (pandas, SciPy, and statsmodels libraries). Continuous variables were assessed for distribution and summarized as mean ± standard deviation (SD) for approximately normally distributed variables and median with interquartile range (IQR) for skewed variables. Age was summarized as mean ± SD and compared between patients with and without IPC using the independent-samples t-test, with the Welch t-test used where appropriate, while BMI, PSA, prostate volume, and PSA density were summarized as median (IQR) and compared using the Mann–Whitney U test.

Categorical variables were presented as frequencies and percentages and compared using Pearson's chi-square test, or Fisher's exact test when expected cell counts were below five. Spearman's rank correlation was used to assess associations between selected continuous variables. Univariable binary logistic regression was performed to estimate crude odds ratios (CORs) with 95% confidence intervals (CIs). Variables with $p < 0.25$ in bivariable analysis, together with clinically relevant variables, were considered for multivariable analysis.

Given the limited number of IPC events (n = 23), the primary multivariable model was restricted to four variables that were selected based on p value and clinical criteria: age (per 10-year increase), hydronephrosis, PSA, and diabetes mellitus. With 23 IPC events and four predictor variables, the model had an events-per-variable (EPV) ratio of 5.75. These variables were selected a priori based on clinical relevance and were entered simultaneously into a multivariable binary logistic regression model. Hydronephrosis and AKI were both markers of obstructive uropathy that were significant on bivariable analysis; however, given their close clinical and mechanistic overlap, hydronephrosis was retained as the more direct anatomical marker of obstruction, while AKI was excluded to avoid redundancy. Hypertension, although significant on bivariable analysis, was not included in the primary model because of the constrained EPV and its comparatively lower biological plausibility as a direct predictor of prostatic malignancy relative to the prespecified variables. Adjusted odds ratios (AORs) with 95% CIs were reported, and multicollinearity among the predictors was assessed using variance inflation factors (VIFs).

Model fit was assessed using the Omnibus Test of Model Coefficients, Hosmer–Lemeshow goodness-of-fit test, and Nagelkerke pseudo-R^2, while discriminatory ability was evaluated using the area under the receiver operating characteristic (ROC) curve (AUC). Two sensitivity analyses were performed to assess the robustness of the primary model: one excluding potentially influential observations identified using Cook's distance, leverage, and studentized residuals, and another replacing raw PSA with its natural logarithm (LnPSA). The consistency of the direction, magnitude, and significance of the associations across the primary and sensitivity models was evaluated to determine the robustness of the findings.

Discriminatory performance was assessed using ROC curve analysis and AUC, with 95% CIs. ROC analyses were also performed for selected individual predictors, including preoperative PSA, PSA density, and age, and sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) were calculated at predefined thresholds. The optimal PSA cut-off was determined using the Youden index. Two-sided $p < 0.05$ was considered statistically significant.

Operational definition

Preoperative acute kidney injury (AKI)

defined according to KDIGO criteria as an increase in serum creatinine of ≥ 0.3 mg/dL within 48 hours or an increase to ≥ 1.5 times the baseline value within the preceding 7 days, documented before surgery (10).

Hypertension

defined as a documented diagnosis of hypertension or current use of antihypertensive medication. If blood pressure measurements were used to define hypertension in the medical records, a blood pressure of ≥ 140/90 mmHg on repeated measurements was considered hypertensive (11).

Diabetes mellitus

defined as a documented diagnosis of diabetes mellitus or current use of glucose-lowering medication. Where laboratory diagnostic criteria were available, diabetes was defined according to standard diagnostic thresholds, including fasting plasma glucose ≥ 126 mg/dL, HbA1c ≥ 6.5%, or random plasma glucose ≥ 200 mg/dL in the presence of classic symptoms of hyperglycemia or hyperglycemic crisis (12).

Hydronephrosis

defined as unilateral or bilateral dilatation of the pelvicalyceal system identified on preoperative ultrasonography (13) .

Prostate volume

Prostate volume was measured by preoperative transabdominal ultrasonography and calculated using the ellipsoid formula (length × width × height × 0.52) (14).

Result

Demographic and Clinical Characteristics

Of 423 patients who underwent TURP or TVP for presumed BPH during the study period, 184 were excluded, leaving 239 for analysis. Of these, 23 (9.6%) were incidentally diagnosed with IPC, while 216 (90.4%) had benign histopathological findings. Of these, 23 patients (9.6%) were incidentally diagnosed with IPC, while 216 (90.4%) had benign histopathological findings. The mean age of the overall study population was 66.7 ± 11.0 years. Patients with IPC were slightly older than those without IPC (69.7 ± 9.8 vs. 66.4 ± 11.0 years), although the difference was not statistically significant (p = 0.159). The largest proportion of patients was aged 60–69 years, while patients with IPC were proportionally more represented in the 70–79-year age group.

Most patients had no history of smoking, and the distributions of diabetes mellitus and cardiac disease did not differ significantly between the IPC and BPH groups. Hypertension was significantly more common among patients with IPC than among those without IPC (47.8% vs. 26.4%, p = 0.030). Lower urinary tract symptoms (LUTS) refractory to medical therapy were the most common indication for surgery in both groups, with no significant difference in the distribution of surgical indications (p = 0.522; Table 1).

Table 1
— Demographic and Clinical Characteristics of Patients Undergoing Surgery for Benign Prostatic Hyperplasia at MCM comprehensive specialized hospital, Addis Ababa, Ethiopia.

Variable	Total (n = 239)	BPH (n = 216)	IPC (n = 23)	P-value
Age, years, mean ± SD	66.68 ± 10.98	66.35 ± 11.03	69.74 ± 9.83	0.159
Age group, n (%)				0.426
<50	10 (4.2)	9 (4.2)	1 (4.3)	
50–59	58 (24.3)	54 (25.0)	4 (17.4)	
60–69	76 (31.8)	71 (32.9)	5 (21.7)	
70–79	59 (24.7)	50 (23.1)	9 (39.1)	
≥80	36 (15.1)	32 (14.8)	4 (17.4)	
BMI, kg/m², Median (IQR)	22(5)	21.5 (4)	22 (5)	0.587
Smoking history, n (%)	23 (9.6)	20 (9.3)	3 (13.0)	0.472
Hypertension, n (%)	68 (28.5)	57 (26.4)	11 (47.8)	0.030
Diabetes mellitus, n (%)	54 (22.7)	46 (21.4)	8 (34.8)	0.145
Cardiac disease, n (%)	22 (9.2)	21 (9.7)	1 (4.3)	0.704
Indication for surgery, n (%)				0.522
LUTS refractory to medical therapy	161 (67.4%)	143 (66.2%)	18 (78.3%)	
Acute urinary retention	64 (26.8%)	60 (27.8%)	4 (17.4%)	
UTI	14 (5.9%)	13 (6.0%)	1 (4.3%)	
BPH, benign prostatic hyperplasia group (patients with benign histopathology, i.e. no IPC); BMI, body mass index; IPC, incidental prostate cancer; IQR, interquartile range; LUTS, lower urinary tract symptoms; SD, standard deviation; UTI, urinary tract infection. Values are n (%) unless otherwise indicated. P-values were calculated using the independent-samples t-test or Mann–Whitney U test for continuous variables and Pearson's chi-square or Fisher's exact test for categorical variables				
Urologic and Laboratory Characteristics				

PSA profiles differed significantly between patients with and without IPC ($p < 0.001$). Among patients with IPC, 8 (34.8%) had a PSA level < 4 ng/mL, 7 (30.4%) had a PSA level of 4–10 ng/mL, and 8 (34.8%) had a PSA level > 10 ng/mL. PSA density was also significantly higher among patients with IPC than among those without IPC, with median values of 0.114 (IQR 0.038–0.286) and 0.035 (IQR 0.022–0.063), respectively ($p < 0.001$).

In contrast, prostate volume did not differ significantly between the groups, with median values of 62.0 (IQR 53.5–79.0) mL among patients with IPC and 65.0 (IQR 55.0–82.0) mL among those without IPC (p

= 0.375). Digital rectal examination findings were likewise comparable between the two groups (p = 0.898).

Hydronephrosis was significantly more common among patients with IPC than among those without IPC (39.1% vs. 14.4%; p = 0.006). Similarly, AKI occurred more frequently in the IPC group than in the non-IPC group (39.1% vs. 8.3%; p < 0.001) (Table 2).

Table 2
Urologic and laboratory characteristics of patients undergoing surgery for benign prostatic hyperplasia at MCM Comprehensive Specialized Hospital, Addis Ababa, Ethiopia.

Variable	**Total (n = 239)**	**BPH (n = 216)**	**IPC (n = 23)**	**P-value**
DRE				0.898
Not reported	196 (82.0)	177 (81.9)	19 (82.6)	
Benign	40 (16.7)	36 (16.7)	4 (17.4)	
Suspicious	2 (0.8)	2 (0.9)	0 (0.0)	
PSA category, n (%)				< 0.001
< 4 ng/mL	172 (72.0)	164 (75.9)	8 (34.8)	
4–10 ng/mL	35 (14.6)	28 (13.0)	7 (30.4)	
> 10 ng/mL	32 (13.4)	24 (11.1)	8 (34.8)	
PSA density, ng/mL/mL, median (IQR)	0.037 (0.023–0.070)	0.035 (0.022–0.063)	0.114 (0.038–0.286)	< 0.001
Prostate volume, mL, median (IQR)	64.0 (55.0–82.0)	65.0 (55.0–82.0)	62.0 (53.5–79.0)	0.375
Hydronephrosis, yes, n (%)	40 (16.7)	31 (14.4)	9 (39.1)	0.006
Acute kidney injury, yes, n (%)	27 (11.3)	18 (8.3)	9 (39.1)	< 0.001

AKI, acute kidney injury; BPH, benign prostatic hyperplasia group (no IPC); DRE, digital rectal examination; IPC, incidental prostate cancer; IQR, interquartile range; PSA, prostate-specific antigen. Values are n (%) unless otherwise indicated. PSA density is PSA divided by prostate volume. P-values were calculated using the Mann–Whitney U test for continuous variables and Pearson's chi-square or Fisher's exact test for categorical variables.

Incidental prostate cancer prevalence and pathological characteristics

Overall, IPC was identified in 23 of 239 patients, corresponding to a prevalence of 9.6%. IPC was identified in 13 of 117 patients undergoing TURP (11.1%) and 10 of 122 patients undergoing TVP (8.2%).

The difference in IPC prevalence between the two surgical procedures was not statistically significant (p = 0.445) (Fig. 1).

Among the 23 patients with IPC, Grade Group 3 was the most frequent pathological grade, occurring in 10 patients (43.5%), followed by Grade Group 2 in 8 patients (34.8%). Grade Group 1 was identified in one patient (4.3%), while Grade Groups 4 and 5 were each identified in two patients (8.7%). Overall, 22 of 23 patients (95.7%) had ISUP Grade Group ≥ 2 (Table 3). Preoperative PSA levels were < 4 ng/mL in 8 patients (34.8%), 4–10 ng/mL in 7 patients (30.4%), and > 10 ng/mL in 8 patients (34.8%). Regarding management, 18 patients (78.3%) received medical androgen deprivation therapy (ADT), three (13.0%) underwent bilateral orchidectomy, and two (8.7%) received ADT with oncology referral (Table 3).

Table 3
Pathological characteristics of patients with incidentally diagnosed prostate cancer

Characteristic	**n (%)**
ISUP grade group	
Grade group 1	1 (4.3)
Grade group 2	8 (34.8)
Grade group 3	10 (43.5)
Grade group 4	2 (8.7)
Grade group 5	2 (8.7)
Preoperative PSA	
≤4 ng/mL	8 (34.8)
4.1–10 ng/mL	7 (30.4)
>10 ng/mL	8 (34.8)
Type of surgery	
Transvesical prostatectomy	10 (43.5)
TURP	13 (56.5)
Management	
Medical androgen deprivation	18 (78.3%)
Bilateral orchidectomy	3 (13.0%)
Androgen deprivation with oncology referral	2 (8.7%)

ADT, androgen deprivation therapy; IPC, incidental prostate cancer; ISUP, International Society of Urological Pathology; PSA, prostate-specific antigen; TURP, transurethral resection of the prostate; TVP,

transvesical prostatectomy. Values are n (%) of the 23 patients with IPC.

IPC, incidental prostate cancer; TURP, transurethral resection of the prostate; TVP, transvesical prostatectomy. Bars show the percentage of patients with IPC after TURP (13/117) and TVP (10/122); the difference was not statistically significant (p = 0.445).

Factors associated with incidental prostate cancer

Univariable logistic regression identified hypertension (p = 0.035), hydronephrosis (p = 0.004), AKI (p < 0.001), preoperative PSA (p < 0.001), and PSA density (p < 0.001) as variables significantly associated with IPC. Variables meeting the prespecified criterion of p < 0.25, together with clinically relevant variables, were considered for multivariable analysis.

In the primary multivariable logistic regression model, age was expressed per 10-year increase, while diabetes mellitus, preoperative PSA, and preoperative hydronephrosis were included as additional predictors. Preoperative PSA, diabetes mellitus, and hydronephrosis remained independently associated with IPC. For each 1 ng/mL increase in preoperative PSA, the odds of IPC increased by approximately 25% (AOR = 1.25, 95% CI: 1.14–1.38; p < 0.001). Diabetes mellitus was also independently associated with IPC (AOR = 5.28, 95% CI: 1.64–16.95; p = 0.005), while preoperative hydronephrosis was associated with nearly fourfold higher odds of IPC (AOR = 3.92, 95% CI: 1.29–11.94; p = 0.016). Age was not independently associated with IPC (AOR = 1.19 per 10-year increase, 95% CI: 0.75–1.88; p = 0.459) (Table 4).

Table 4
Factors associated with incidental prostate cancer

Variable	Crude OR (95% CI)	P-value	Adjusted OR (95% CI)	P-value
Age, per 10-year increase	1.33 (0.89–1.98)	**0.160**	1.19 (0.75–1.88)	0.459
Preoperative PSA, per 1 ng/mL increase	1.223 (1.132–1.322)	**< 0.001**	1.25 (1.14–1.38)	**< 0.001**
Diabetes mellitus	1.96 (0.78–4.91)	**0.151**	5.28 (1.64–16.95)	**0.005**
Hydronephrosis	3.84 (1.53–9.62)	**0.004**	3.92 (1.29–11.94)	**0.016**
Hypertension	2.56 (1.07–6.12)	**0.035**		
AKI	7.07 (2.69–18.59)	**< 0.001**		
BMI, per 1 kg/m² increase	1.04 (0.93–1.17)	0.517		
Smoking history	1.47 (0.40–5.38)	0.561		
Cardiac disease	0.42 (0.05–3.29)	0.410		
Indication for surgery		0.509		
LUTS refractory to medical therapy	1.64 (0.20–13.26)	0.645		
Acute urinary retention	0.87 (0.09–8.40)	0.902		
DRE finding	0.898 (0.308–2.616)	0.843		
Prostate volume, per 1 mL increase	0.99 (0.97–1.01)	0.493		
Type of surgical procedure	1.400 (0.589–3.330)	0.447		

AKI, acute kidney injury; BMI, body mass index; CI, confidence interval; DRE, digital rectal examination; IPC, incidental prostate cancer; LUTS, lower urinary tract symptoms; OR, odds ratio; PSA, prostate-specific antigen. P-values are from logistic regression. The adjusted model included age, preoperative PSA, diabetes mellitus, and hydronephrosis; blank cells indicate variables not entered in the multivariable model. The reference category is absence of the characteristic for binary variables.

Model performance

The primary multivariable logistic regression model demonstrated a statistically significant improvement over the intercept-only model (Omnibus $\chi^2 = 43.792$, df = 4, $p < 0.001$). The model had a − 2 log likelihood of 107.402, with a Nagelkerke R^2 of 0.357.

The Hosmer–Lemeshow test showed no evidence of poor model fit ($\chi^2 = 11.759$, df = 8, p = 0.162). The model demonstrated good discriminatory ability, with an AUC of 0.838 (95% CI: 0.753–0.923; p < 0.001). At a predicted-probability threshold of 0.50, specificity was 99.1%, sensitivity was 34.8%, and overall classification accuracy was 92.9%. Variance inflation factors ranged from 1.018 to 1.052, indicating no evidence of problematic multicollinearity (Table 5).

Table 5
Model fitness and performance of the final multivariable logistic regression model

Measure	Value
Omnibus test	$\chi^2 = 43.792$, df = 4, $p < 0.001$
−2 Log Likelihood	107.402
Nagelkerke R^2	0.357
Hosmer–Lemeshow test	$\chi^2 = 11.759$, df = 8, $p = 0.162$
AUC (95% CI)	0.838 (0.753–0.923)
Sensitivity	34.8%
Specificity	99.1%
Overall classification accuracy	92.9%
VIF range	1.018–1.052

AUC, area under the receiver operating characteristic curve; CI, confidence interval; VIF, variance inflation factor. Sensitivity, specificity, and overall classification accuracy are at a predicted-probability threshold of 0.50.

Sensitivity analyses

The sensitivity analyses were performed to assess the robustness of the primary multivariable findings. The first evaluated whether the results were dependent on the functional form of PSA, while the second assessed the effect of excluding potentially influential observations.

In the first sensitivity analysis, raw preoperative PSA was replaced with its natural logarithmic transformation (LnPSA), while age, diabetes mellitus, and preoperative hydronephrosis were retained. The principal findings remained consistent. Age remained non-significant (AOR = 1.24, 95% CI: 0.81–1.89; p = 0.332), whereas diabetes mellitus (AOR = 4.64, 95% CI: 1.52–14.19; p = 0.007), LnPSA (AOR = 3.73, 95% CI: 2.03–6.86; p < 0.001), and hydronephrosis (AOR = 3.72, 95% CI: 1.30–10.62; p = 0.014) remained significantly associated with IPC. Model discrimination remained good (AUC = 0.817, 95% CI: 0.731–0.903), and the Hosmer–Lemeshow test indicated adequate calibration (p = 0.274).

In the second sensitivity analysis, three observations identified as potentially influential based on Cook's distance, leverage, and studentized residuals were temporarily excluded. After refitting the primary model, the findings remained substantively unchanged. Age remained non-significant (AOR = 1.38, 95% CI: 0.83–2.29; p = 0.211), while diabetes mellitus (AOR = 4.23, 95% CI: 1.19–15.13; p = 0.026), raw preoperative PSA (AOR = 1.26 per 1 ng/mL increase, 95% CI: 1.14–1.39; p < 0.001), and hydronephrosis (AOR = 3.98, 95% CI: 1.19–13.33; p = 0.025) remained statistically significant. Model discrimination was maintained (AUC = 0.851, 95% CI: 0.759–0.942), with no evidence of poor calibration (p = 0.371).

Overall, the direction and statistical significance of the principal associations remained consistent across both sensitivity analyses, supporting the robustness of the primary findings. In particular, the association of preoperative PSA with IPC remained highly stable, while diabetes mellitus and hydronephrosis remained significant across the alternative model specifications. Age remained non-significant across all models (Table 6).

Table 6
Sensitivity analyses of factors independently associated with IPC

Predictor	Primary model AOR (95% CI)	Sensitivity 1: Influential observations excluded AOR (95% CI)	Sensitivity 2: LnPSA AOR (95% CI)
Age, per 10-year increase	1.19 (0.75–1.88)	1.38 (0.83–2.29)	1.24 (0.81–1.89)
Diabetes mellitus	5.28 (1.64–16.95)	4.23 (1.19–15.13)	4.64 (1.52–14.19)
Preoperative PSA, per 1 ng/mL	1.25 (1.14–1.38)	1.26 (1.14–1.39)	—
LnPSA	—	—	3.73 (2.03–6.86)
Preoperative hydronephrosis	3.92 (1.29–11.94)	3.98 (1.19–13.33)	3.72 (1.30–10.62)

AOR, adjusted odds ratio; CI, confidence interval; LnPSA, natural logarithm of PSA; PSA, prostate-specific antigen. Primary model: age, diabetes mellitus, preoperative PSA, and hydronephrosis. Sensitivity 1: PSA replaced by LnPSA. Sensitivity 2: three influential observations excluded. —, variable not included in that model.

Diagnostic performance of preoperative markers for incidental prostate cancer

Receiver operating characteristic (ROC) curve analysis of the multivariable predictive model for incidental prostate cancer (IPC) demonstrated good discriminatory ability, with an area under the curve (AUC) of 0.838 (95% CI: 0.753–0.923; p < 0.001) (Fig. 2). Among individual predictors, preoperative PSA and PSA density showed comparable discriminatory performance, both outperforming patient age as a standalone predictor (Fig. 2).

NPVs ranged from 92.8% to 95.3% across all thresholds, only modestly above the baseline NPV of 90.4% expected from the overall IPC prevalence of 9.6%, indicating that none of the tested cut-offs reliably excluded IPC when negative (Table 7).

AUC, area under the curve; IPC, incidental prostate cancer; PSA, prostate-specific antigen. ROC curves are shown for age, preoperative PSA (ng/mL), and PSA density; diagonal segments result from tied values.

Table 7
Performance of preoperative markers and thresholds for detecting incidental prostate cancer

Threshold	TP	FN	FP	TN	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
PSA > 4 ng/mL	15	8	52	164	65.2	75.9	22.4	95.3
PSA > 6 ng/mL	13	10	31	185	56.5	85.6	29.5	94.9
PSA > 10 ng/mL	8	15	24	192	34.8	88.9	25.0	92.8
PSA density > 0.15	11	12	21	195	47.8	90.3	34.4	94.2

TP, true positive; FN, false negative; FP, false positive; TN, true negative; PPV, positive predictive value; NPV, negative predictive value. PSA is in ng/mL and PSA density in ng/mL/mL. Sensitivity, specificity, PPV, and NPV compare the 23 IPC cases with the 216 patients with benign histology.

Management of Incidentally Diagnosed Prostate Cancer According to ISUP Grade Group

Regarding management, 18 patients (78.3%) received medical ADT, three (13.0%) underwent surgical ADT through bilateral orchidectomy, and two (8.7%) received ADT with oncology referral. Medical ADT was used in all patients with Grade Group 1 disease (1/1, 100.0%), 16 of 18 patients (88.9%) with Grade Group 2–3 disease, and one of four patients (25.0%) with Grade Group 4–5 disease. Surgical ADT was performed in three of four patients (75.0%) with Grade Group 4–5 disease (Table 8).

Table 8
Management of incidentally diagnosed prostate cancer according to ISUP Grade Group risk category

Gleason/ISUP risk group	Medical ADT	Surgical ADT	ADT + oncology referral	Total
Low grade (GG1)	**1 (100.0%)**	**0 (0%)**	**0 (0%)**	**1**
Intermediate grade (GG2–3)	**16 (88.9%)**	**0 (0%)**	**2 (11.1%)**	**18**
High grade (GG4–5)	**1 (25.0%)**	**3 (75.0%)**	**0 (0%)**	**4**
Total	**18 (78.3%)**	**3 (13.0%)**	**2 (8.7%)**	**23**

ADT, androgen deprivation therapy; GG, ISUP Grade Group; ISUP, International Society of Urological Pathology. Surgical ADT was bilateral orchidectomy. Percentages are row percentages.

Correlation analyses

Spearman correlation analysis demonstrated a statistically significant but weak positive association between age and preoperative PSA level, indicating that older age was associated with slightly higher PSA concentrations. In contrast, no association was observed between age and prostate volume. BMI was not significantly associated with either preoperative PSA or prostate volume. The correlation between BMI and PSA was very weak and inverse, whereas the correlation between BMI and prostate volume was very weak and positive. Among the 23 patients with IPC, preoperative PSA showed a very weak positive correlation with ISUP Grade Group (ρ = 0.086, p = 0.698; n = 23). This association was not statistically significant (Table 9).

Table 9
Spearman correlation analysis of demographic, biochemical, and urologic variables

Variable pair	Spearman's ρ	P-value
Age vs. preoperative PSA	0.147	0.023
Age vs. prostate volume	−0.001	0.987
BMI vs. preoperative PSA	−0.093	0.151
BMI vs. prostate volume	0.081	0.209
Preoperative PSA vs. ISUP Grade Group*	0.086	0.698

BMI, body mass index; IPC, incidental prostate cancer; ISUP, International Society of Urological Pathology; PSA, prostate-specific antigen; ρ, Spearman's rank correlation coefficient. *Calculated among the 23 patients with IPC only.

Discussion

In this retrospective cohort of 239 men who underwent surgery for presumed BPH at MCM Addis Ababa, IPC was found in 23 (9.6%), and 22 of these 23 (95.7%) were ISUP Grade Group ≥ 2. In the primary multivariable model, higher preoperative PSA (AOR 1.25 per ng/mL, 95% CI 1.14–1.38), diabetes mellitus (AOR 5.28, 95% CI 1.64–16.95) and preoperative hydronephrosis (AOR 3.92, 95% CI 1.29–11.94) were independently associated with IPC, while age was not. The model discriminated well (AUC 0.838). The PSA association was stable in both sensitivity analyses. PSA and PSA density showed comparable discrimination, and PSA density > 0.15 ng/mL/mL gave 90.3% specificity but only 47.8% sensitivity. Notably, 34.8% of IPC cases had a preoperative PSA below 4 ng/mL..

The prevalence of IPC in our cohort was broadly comparable to international estimates. It was similar to the 10.7% reported in a Pakistani TURP series and close to the pooled prevalence of approximately 10% in a meta-analysis of patients undergoing TURP for presumed BPH (15, 16). A recent TURP cohort reported a prevalence of 8.4%, further supporting the position of our estimate within the contemporary

range (17). Prevalence did not differ significantly between TURP (11.1%) and transvesical prostatectomy (8.2%).

In contrast, IPC prevalence was substantially higher in a Somali study (17.6%) and lower in a US national dataset (2.2%).(18).These differences may reflect variation in preoperative PSA screening and biopsy practices, histopathological sampling, and differences in the characteristics of men undergoing surgery for BPH. Regional differences in healthcare access and thresholds for surgical intervention may also contribute (19). Our estimate therefore lies toward the middle-to-lower end of the published range and is closer to contemporary estimates from well-resourced settings than to the Somali estimate, despite the shared resource-limited context.

Preoperative PSA was a strong and independent predictor of IPC in our cohort. The association was robust, with an adjusted odds ratio of approximately 1.25 per ng/mL, and results were similar in sensitivity analyses using log-transformed PSA and after exclusion of influential observations. PSA density was not entered into the multivariable model or the sensitivity analyses, but it was significantly higher in men with IPC (median 0.114 vs 0.035 ng/mL/mL) and was evaluated in ROC and threshold analyses. There it showed discrimination comparable to PSA and better than age. A PSA density threshold of > 0.15 ng/mL/mL achieved 90.3% specificity and a PPV of 34.4%, although its sensitivity was 47.8%.

These findings are consistent with recent evidence. An earlier meta-analysis of 23 studies identified higher PSA as the only independent predictor of IPC after BPH surgery, whereas age and prostate volume were not predictive (15). A 2025 systematic review and meta-analysis of 21 studies involving 10,842 patients found significant associations between IPC and both preoperative PSA and PSA density (20). A more recent meta-analysis of 25 studies involving 247,966 patients likewise identified elevated PSA and PSA density > 0.15 ng/mL/cc as significant risk factors for IPC (8). A recent TURP cohort with an IPC prevalence of 8.4% found elevated PSA density to be independently associated with IPC and suggested that it may be particularly useful among patients with borderline PSA levels (17).

Taken together, these findings support considering PSA density alongside PSA in preoperative risk assessment before BPH surgery. This conclusion should be drawn cautiously, because PSA density was not formally compared with PSA in our multivariable analysis, and one case-control study in men with large prostates found PSA density alone unreliable for predicting IPC (21). Incorporating PSA density may nevertheless be feasible in our setting, because transrectal ultrasonography is already used to estimate prostate volume, allowing PSA density to be calculated without an additional diagnostic test.

An important clinical finding was that 8 of 23 patients with IPC (34.8%) had a preoperative PSA level < 4 ng/mL. Thus, reliance on PSA alone would have failed to identify approximately one-third of IPC cases in our cohort. This finding reinforces the limitation of PSA as a standalone marker for occult prostate cancer; incidental cancers have also been documented among men with PSA levels below conventional thresholds (22). Therefore, a normal PSA should not be considered sufficient grounds to omit or reduce the thorough histopathological examination of tissue obtained during TURP or TVP. Complete or

adequately extensive pathological examination remains important because clinically unsuspected cancers may be present despite apparently reassuring preoperative PSA values (23).

Hydronephrosis and AKI were significantly associated with IPC in our cohort, with hydronephrosis remaining independently associated after adjustment (AOR 3.92). These findings are less commonly evaluated in IPC literature and may represent useful signals in resource-limited settings where patients present with more advanced obstruction. This may reflect tumor-related urinary tract compromise or, alternatively, greater overall disease severity at presentation. However, this association should be considered hypothesis-generating and requires confirmation in larger cohorts.

Diabetes mellitus was unexpectedly associated with IPC (AOR 5.28), with a similar association across sensitivity analyses (AOR 4.64 and 4.23). This contrasts with evidence suggesting an inverse association between diabetes and prostate cancer, including a recent systematic review and meta-analysis reporting a pooled OR of 0.68 and a population-based study reporting an adjusted OR of 0.64 (24, 25). Greater healthcare contact and more frequent PSA testing among diabetic patients may partly explain our finding. However, the small number of diabetic patients with IPC (8/23) and the wide confidence interval (1.64–16.95) indicate substantial uncertainty, and this association should therefore be considered hypothesis-generating.

Age was not independently associated with IPC in our cohort (AOR 1.19 per decade), remaining non-significant in the primary and sensitivity models. Although age is frequently reported as a risk factor for prostate cancer, a meta-analysis of IPC after BPH surgery found no significant association between age and IPC (OR 1.02, p = 0.48), supporting our finding (15). This may reflect the relatively restricted age range of men undergoing BPH surgery or mediation through PSA, with age-related risk partly captured by increasing PSA levels.

Age showed a weak but significant positive correlation with PSA (ρ = 0.147), consistent with established evidence that PSA tends to increase with age (26). In contrast, age was essentially unrelated to prostate volume (ρ=−0.001), differing from studies reporting positive associations, potentially reflecting differences in population composition. BMI showed weak, non-significant correlations with PSA and prostate volume, consistent with the mixed evidence regarding these relationships (27). Finally, PSA was not significantly correlated with ISUP Grade Group among IPC patients (ρ = 0.086, n = 23); this should be interpreted cautiously because the small subgroup substantially limits statistical power rather than as evidence of no relationship.

A striking finding was the high proportion of clinically significant disease among patients with IPC: 22 of 23 cases (95.7%) were Grade Group ≥ 2. This was considerably higher than the 49% of Pakistani IPC cases reported to have Gleason scores > 7, although direct comparison is limited by differences in grading definitions and study populations (16). Despite this substantial burden, none of our patients received radical prostatectomy, radiotherapy, or active surveillance. Management was almost uniformly ADT-based and showed little differentiation by grade; the one patient with Grade Group 1 disease also received ADT, which is not consistent with guideline-recommended active surveillance. No staging

investigations or structured follow-up were documented. This contrasts with contemporary US data, where approximately 68% of IPC patients received no active treatment, but definitive local treatment remained available to a proportion of patients (18). The more limited treatment pattern observed in our cohort may therefore reflect constraints in access to staging imaging, radiotherapy, surgical oncology services, and structured urologic oncology pathways rather than clinical judgment alone. These findings highlight the need for structured protocols for staging, risk stratification, referral, treatment, and follow-up of IPC in similar resource-limited settings.

Conclusion

In this single-center cohort, IPC was identified in 9.6% of patients undergoing TURP or TVP for presumed BPH. Preoperative PSA, diabetes mellitus, and hydronephrosis were independently associated with IPC. PSA density was also higher in patients with IPC and showed useful specificity at a threshold of > 0.15 ng/mL/mL, but it was not included in the multivariable model. About one-third of IPC cases had a PSA below 4 ng/mL, so a normal PSA should not reduce the thoroughness of histopathological examination of resected tissue. Most incidental cancers were clinically significant (ISUP Grade Group ≥ 2). Yet management was uniformly ADT-based, and no patient had documented staging, definitive local therapy, or structured follow-up. These findings are hypothesis-generating given the single-center, retrospective design and the small number of events. They suggest PSA density merits evaluation in larger studies and show a need for structured post-diagnosis care pathways for IPC in similar resource-limited settings.

Strength

This study benefits from a comprehensive dataset spanning sociodemographic, clinical, laboratory, radiological, and histopathological domains, allowing multivariable adjustment and a series of sensitivity analyses (functional-form testing of PSA and exclusion of influential observations) that are not universally reported in comparable single-center IPC studies. To our knowledge, this is among the few studies in an East African setting to report not only IPC prevalence and predictors but also post-diagnosis management patterns, offering insight into a stage of the care pathway rarely captured in similar literature.

Limitation

Several limitations should be acknowledged. First, this was a single-center, retrospective study, which may limit generalizability to other settings. Second, the small number of IPC events (n = 23) yielded a limited events-per-variable ratio (5.75) in the multivariable model, restricting the number of covariates that could be reliably adjusted for and producing wide confidence intervals around several estimates, particularly for diabetes mellitus. Third, formal staging data (e.g., imaging-based local or metastatic staging) were not consistently available, precluding assessment of disease extent beyond histopathological grade. Fourth, retrospective ascertainment of management plans relied on chart documentation. Finally, no long-term follow-up or oncologic outcome data were available, limiting our

ability to comment on survival or disease progression. These findings should therefore be interpreted as hypothesis-generating, warranting confirmation in larger, prospective, multi-center cohorts.

The extent of tumor (T1a vs. T1b), the number of positive chips, and the individual Gleason patterns were not consistently reported, which limited pathological characterization beyond the ISUP Grade Group.

Abbreviations

Acute Kidney Injury (AKI), Adjusted Odds Ratio (AOR), Androgen Deprivation Therapy (ADT), Area Under the Curve (AUC), Benign Prostatic Hyperplasia (BPH), Body Mass Index (BMI), Confidence Interval (CI), Crude Odds Ratio (COR), Digital Rectal Examination (DRE), Events Per Variable (EPV), False Negative (FN), False Positive (FP), Grade Group (GG), Incidental Prostate Cancer (IPC), Institutional Review Board (IRB), Interquartile Range (IQR), International Society of Urological Pathology (ISUP), Lower Urinary Tract Symptoms (LUTS), Natural Logarithm of Prostate-Specific Antigen (LnPSA), Negative Predictive Value (NPV), Odds Ratio (OR), Positive Predictive Value (PPV), Prostate-Specific Antigen (PSA), Receiver Operating Characteristic (ROC), Standard Deviation (SD), Transurethral Resection of the Prostate (TURP), Transvesical Prostatectomy (TVP), True Negative (TN), True Positive (TP), Urinary Tract Infection (UTI), Variance Inflation Factor (VIF).

Declarations

Competing interests

The authors declare that they have no competing interests.

Ethical Concerns

Ethical approval was obtained from the Institutional Review Board (IRB) of MCM Comprehensive Specialized Hospital (PRO-140126). Beneficence was ensured and non-maleficence was upheld because the study involved retrospective review of patient records without direct patient contact. Autonomy was respected through an IRB-approved waiver of informed consent. Confidentiality was maintained by anonymizing all patient data and restricting data access to the research team only.

2.Availability of data and materials

The datasets generated and/or analyzed during the current study are not publicly available due to institutional and patient confidentiality requirements but may be available from the corresponding author upon reasonable request and subject to appropriate institutional approval.

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Author contribution

ET conceptualized the study, contributed to its design, and critically reviewed and revised the manuscript. **MB** co-led the study as second principal investigator, contributed to the study design, developed the methodology, supervised the research process, performed the statistical analysis and interpretation of the data, and prepared the first draft of the manuscript. **DL** contributed to the methodology, performed data analysis and interpretation, drafting and revising the manuscript. **KF**, **CE**, and **BA** contributed to data collection and critically reviewed the manuscript for important intellectual content. **AZ** reviewed the study methodology and critically revised the manuscript for important intellectual content. **SD** provided senior oversight, critically reviewed the manuscript, and contributed to writing and refining the conclusion. All authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

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STROBE guidelines

This study was reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. The completed STROBE checklist is provided as a supplementary file.

Clinical trial number

Not applicable.

References

1. Bray F, Laversanne M, Sung H, Ferlay J, Siegel RL, Soerjomataram I, et al. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin. 2024;74(3):229–63.
2. Gizaw M, Parkin DM, Asmare S, Lukas K, Getachew S, Destaw A, et al. Cancer incidence in Addis Ababa, 2018–2022: The only population based cancer registry in Ethiopia. Cancer Epidemiol. 2026;102:103048.
3. Lee SWH, Chan EMC, Lai YK. The global burden of lower urinary tract symptoms suggestive of benign prostatic hyperplasia: A systematic review and meta-analysis. Sci Rep. 2017;7(1):7984.
4. The global. regional, and national burden of benign prostatic hyperplasia in 204 countries and territories from 2000 to 2019: a systematic analysis for the Global Burden of Disease Study 2019.

Lancet Healthy Longev. 2022;3(11):e754–76.

5. Netto GJ, Epstein JI. Benign Mimickers of Prostate Adenocarcinoma on Needle Biopsy and Transurethral Resection. Surg Pathol Clin. 2008;1(1):1–41.
6. Otto B, Barbieri C, Lee R, Te AE, Kaplan SA, Robinson B, et al. Incidental prostate cancer in transurethral resection of the prostate specimens in the modern era. Adv Urol. 2014;2014:627290.
7. Cuzick JM, Stone S, Lenz L, Flake DD, Rajamani S, Moller H, et al. Validation of the cell cycle progression score to differentiate indolent from aggressive prostate cancer in men diagnosed through transurethral resection of the prostate biopsy. Cancer Rep (Hoboken). 2022;5(8):e1535.
8. Cormio A, Matsukawa A, Dematteis A, Alfarhan AR, Miszczyk M, Roessler N et al. Risk factors for incidental prostate cancer following surgery for benign prostatic enlargement: A systematic review and meta-analysis. Prostate Cancer Prostatic Dis. 2026.
9. Mohamed AH, Abdullahi IM, Warsame FF, Mohamud HA. Incidence and associated factors for incidental prostate cancer among patients who underwent surgery for benign prostatic hyperplasia: first report from Somalia. J Cancer Res Clin Oncol. 2023;149(7):4041–6.
10. Matuszkiewicz-Rowińska J, Małyszko J. Acute kidney injury, its definition, and treatment in adults: guidelines and reality. Pol Arch Intern Med. 2020;130(12):1074–80.
11. Gabb GM, Mangoni AA, Anderson CS, Cowley D, Dowden JS, Golledge J, et al. Guideline for the diagnosis and management of hypertension in adults – 2016. Med J Aust. 2016;205(2):85–9.
12. 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes-2024. Diabetes Care. 2024;47(Suppl 1):S20–42.
13. Laquerre J, Hydronephrosis. Diagnosis, Grading, and Treatment. Radiol Technol. 2020;92(2):135–51.
14. Rodriguez E Jr., Skarecky D, Narula N, Ahlering TE. Prostate volume estimation using the ellipsoid formula consistently underestimates actual gland size. J Urol. 2008;179(2):501–3.
15. Guo Z, He J, Huang L, Wang Z, Hu P, Wang S, et al. Prevalence and risk factors of incidental prostate cancer in certain surgeries for benign prostatic hyperplasia: A systematic review and meta-analysis. Int Braz J Urol. 2022;48(6):915–29.
16. Janjua TK, Yousuf MA, Iqbal MT, Memon SM, Abdullah A, Faridi N, et al. Incidental finding of prostate cancer in Transurethral Resection of Prostate (TURP) specimens: a retrospective analysis from a Tertiary Care Hospital in Pakistan. Pan Afr Med J. 2021;39:20.
17. Ghazwani Y, Alhaider A, Aldharab R, Aldumiaikhi F, Albogami N, Alathel A, et al. Incidence of prostate adenocarcinoma in transurethral resection of the prostate patients. Urol Ann. 2025;17(1):43–7.
18. Bologna E, Licari LC, Franco A, Ditonno F, Manfredi C, De Nunzio C et al. Incidental Prostate Cancer in Patients Treated for Benign Prostatic Hyperplasia: Analysis from a Contemporary National Dataset. Diagnostics (Basel). 2024;14(7).
19. Culp MB, Soerjomataram I, Efstathiou JA, Bray F, Jemal A. Recent Global Patterns in Prostate Cancer Incidence and Mortality Rates. Eur Urol. 2020;77(1):38–52.

20. Wang Y, Li X, Yang H, Yin C, Wu Y, Chen X. Predictive factors of incidental prostate cancer in patients undergoing surgery for presumed benign prostatic hyperplasia: an updated systematic review and meta-analysis. Front Oncol. 2025;15:1561675.
21. Farzat M, Wagenlehner FM. Incidental prostate cancer: is prostate-specific antigen density a valid predictor? A case-control study. Ther Adv Urol. 2025;17:17562872251386996.
22. Froehner M, Buck LM, Koch R, Hakenberg OW, Wirth MP. Derivatives of prostate-specific antigen as predictors of incidental prostate cancer. BJU Int. 2009;104(1):25–8.
23. Rohr LR. Incidental adenocarcinoma in transurethral resections of the prostate. Partial versus complete microscopic examination. Am J Surg Pathol. 1987;11(1):53–8.
24. Drab A, Wdowiak K, Kanadys W, Zajączkowski K, Koczkodaj P, Religioni U et al. Diabetes Mellitus and Prostate Cancer Risk-A Systematic Review and Meta-Analysis. Cancers (Basel). 2024;16(23).
25. Coker AL, Sanderson M, Zheng W, Fadden MK. Diabetes mellitus and prostate cancer risk among older men: population-based case-control study. Br J Cancer. 2004;90(11):2171–5.
26. Pinsky PF, Kramer BS, Crawford ED, Grubb RL, Urban DA, Andriole GL, et al. Prostate volume and prostate-specific antigen levels in men enrolled in a large screening trial. Urology. 2006;68(2):352–6.
27. Ochiai A, Fritsche HA, Babaian RJ. Influence of anthropometric measurements, age, and prostate volume on prostate-specific antigen levels in men with a low risk of prostate cancer. Urology. 2005;66(4):819–23.

Figures

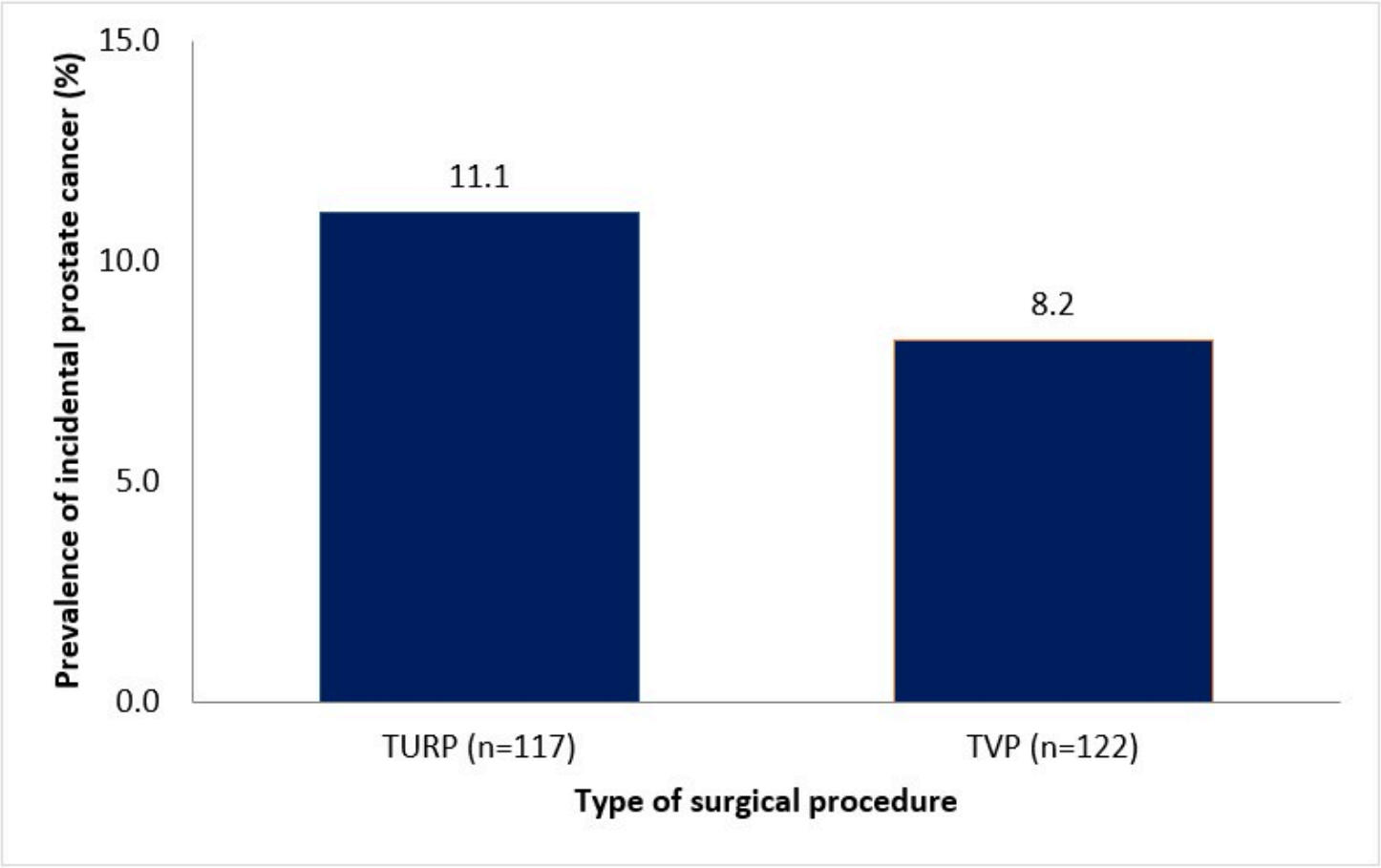


Figure 1

Prevalence of incidental prostate cancer according to type of surgical procedure

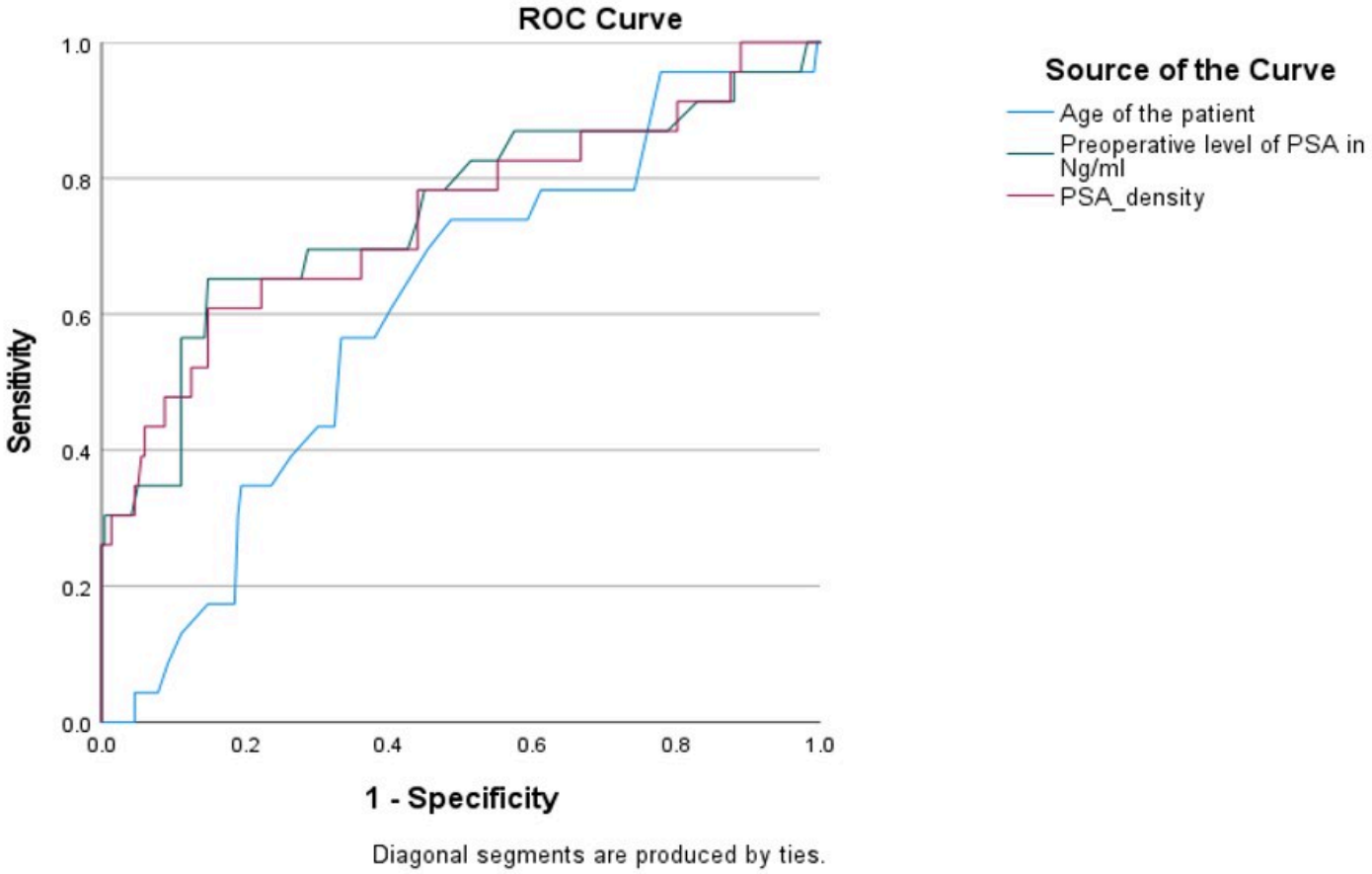


Figure 2

Receiver operating characteristic (ROC) curves of age, preoperative PSA, and PSA density for detecting incidentally diagnosed prostate cancer.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- STROBEchecklistcompleted1.docx