DOI: 10.4081/aiua.2026.14669

ORIGINAL PAPER

Incidental prostate cancer following surgery for benign prostatic hyperplasia: A cohort study

Rui Pedrosa, João Lorigo, Ana João Guerra, Bárbara Figueiredo, Vasco Quaresma, Miguel Eliseu, Paulo Temido, Arnaldo Figueiredo

Department of Urology and Renal Transplantation, ULS Coimbra, Coimbra, Portugal.

Summary

Background: *Incidental prostate cancer (iPCa) remains a clinically relevant diagnosis in men undergoing surgery for benign prostatic hyperplasia (BPH). This study aimed to determine the incidence, characterize pathological features, and identify preoperative predictors of iPCa.*

Materials and methods: *We conducted a retrospective cohort study of 735 men undergoing BPH surgery between November 2020 and December 2024. Demographic, clinical, surgical, and pathological variables were analyzed. Predictors of iPCa were evaluated using logistic regression; discrimination was assessed by ROC curves and Youden-optimized cut-offs.*

Results: *The incidence of iPCa was 5.6%. Among iPCa cases, 48.8% were ISUP 1, while 17.1% corresponded to high-grade tumors (ISUP ≥ 4). Patients with iPCa had significantly higher PSA (4.8 vs 1.9 ng/mL), higher PSA density (PSAD 0.099 vs 0.029 ng/mL/cm³), and smaller prostates (47 vs 66 mL) (all p < 0.001). In multivariable analysis including age and PSAD, only PSAD remained independently associated with iPCa (per 0.01 ng/mL/cm³ increase). PSAD discriminated iPCa better than PSA (AUC 0.86 vs 0.80). A PSAD threshold around 0.15 ng/mL/cm³ provided balanced performance (sensitivity ≈0.82; specificity ≈0.78). At a median follow-up of 24 months, most patients were managed conservatively (active surveillance or watchful waiting) with favorable short-term biochemical control; a minority required systemic therapy (hormonotherapy), and cancer-specific mortality was low.*

Conclusions: *iPCa occurred infrequently after BPH surgery, although higher-grade tumors were still observed. PSA density was the strongest preoperative predictor and should be integrated into risk stratification before BPH surgery.*

KEY WORDS: *Prostatic hyperplasia; Surgery; Prostatic neoplasms; Incidental findings; Diagnosis.*

Submitted 28 November 2025; Accepted 6 December 2025

INTRODUCTION

Benign prostatic hyperplasia (BPH) is highly prevalent condition, affecting over half of men > 60 years (1). While most patients are managed conservatively, 20-30% with moderate-severe lower urinary tract symptoms ultimately require surgery when medical therapy fails (2, 3).

Additional surgical indications include recurrent acute urinary retention and persistently elevated post-void residuals. Patients submitted to surgical interventions for BPH such as *transurethral resection of prostate* (TURP) or *simple prostatectomy* (SP) are usually evaluated prior to treatment to rule out malignancy that could change the therapeutic strategy (3).

Incidental prostate cancer (iPCa) refers to cancer unexpectedly identified on histopathological assessment of tissue resected for presumed BPH (2). According to the TNM classification, tumors involving ≤ 5% of resected tissue are staged pT1a, and those > 5% as pT1b (6). The pooled incidence of iPCa after BPH surgery is approximately 8.8%, with nearly 20% of these being clinically significant disease (ISUP Grade Group ≥ 2; Gleason ≥ 3+4) (4). Although many iPCa cases are low volume and indolent, a relevant subset warrants definitive oncologic treatment (2, 5, 7).

This study primarily aims to estimate the incidence of iPCa among men undergoing surgery for benign prostatic hyperplasia and to describe their demographic, clinical, and pathological characteristics. Secondary aims are to analyze preoperative prostate cancer screening and relevant perioperative factors; identify preoperative predictors of iPCa – focusing on PSA, prostate volume, and PSA density; and characterize initial post-diagnosis management and short-term oncologic outcomes.

MATERIALS AND METHODS

This single-center, retrospective observational study evaluated the incidence of iPCa after surgery for BPH.

Setting and population

We screened all consecutive patients who underwent BPH surgery between November 2020 and December 2024. Two surgical approaches were analyzed according to our institutional practice at the time: TURP for prostates ≤ 80 cm³ and SP for prostates > 80 cm³.

Eligibility criteria

Of 820 operated patients (568 TURP and 252 SP), we excluded 85 cases: 62 with a previous prostate cancer diagnosis and 23 who underwent other procedures (transurethral incision of the prostate or bladder neck incision). The final cohort comprised 735 patients. iPCa was defined as adenocarcinoma identified on histopathological assessment of tissue resected for presumed BPH in patients without a prior prostate cancer diagnosis.

Data collection

We retrospectively extracted demographic, clinical, laboratory, imaging, surgical, and pathological data from electronic records. Variables included were, when available, age, PSA, prostate volume, PSA density, digital rectal examination, prior negative biopsy, use of 5-alpha-reductase inhibitors, urinary retention/catheterization, perioperative details, and pathology (ISUP Grade Group, pT1a/pT1b according to the percentage of tumor in the resected specimen).

Ethics

As a retrospective chart review of anonymized data with no patient contact or intervention, the requirement for individual informed consent was waived in accordance with institutional and national regulations. All data were handled confidentially, and the study adhered to the principles of the Declaration of Helsinki (2024 update).

Statistical analysis

Continuous variables are presented as median and interquartile range and categorical variables as counts (percentages). Group comparisons used the Mann-Whitney U test.

Preoperative predictors of iPCa were assessed using logistic regression, reporting *odds ratios* (OR) with 95% *confidence intervals* (CI). Discriminative performance was evaluated with *receiver operating characteristic* (ROC) curves and *area under the curve* (AUC); optimal cut-offs for PSA and PSA density were obtained using the Youden index, with corresponding sensitivity and specificity. Two-sided p-values < 0.05 were considered statistically significant. Analyses were performed with standard statistical software.

Results

Demographic and preoperative assessment

Over the 49-month study period (November 2020 to December 2024), 41 of 735 surgically treated patients (5.6%) were found to have an iPCa on histopathology. As shown in Table 1, the mean age at diagnosis was 73 years (range, 48-90). Regarding comorbidities, arterial hypertension was the most prevalent condition, affecting 82.9% of patients, followed by dyslipidemia (78.0%) and diabetes mellitus (48.8%). With respect to prior medical therapy for BPH, 17 patients were receiving combination treatment with an alpha-adrenergic receptor antagonist and a 5-alpha-reductase inhibitor at the time of surgery, reflecting guideline-based management of moderate-to-severe LUTS. Alpha-blocker monotherapy was the second most frequently prescribed regimen (10 patients), whereas 5-alpha-reductase inhibitor monotherapy and other combinations were less common; 7 patients had received no medical therapy.

As part of the standard preoperative work-up, all patients underwent serum prostate-specific antigen (PSA) testing. The mean preoperative PSA was 3.06 ng/mL (range, 0.10-27.90 ng/mL). Ultrasound-based assessment of prostate volume showed a mean of 45 cm³ (range, 20-197 cm³), allowing calculation of PSA density, which yielded a mean of 0.064 ng/mL/cm³ (range, 0.003-0.636 ng/mL/cm³). *Multiparametric prostate MRI* (mpMRI) and preoperative prostate biopsy were performed selectively in patients with clinical suspicion of malignancy-namely, concerning PSA kinetics, abnormal digital rectal examination, or imaging findings. Overall, 2 patients (4.9%) underwent mpMRI (one with PIRADS 3 and another with PIRADS 4) and 3 (7.3%) were submitted to prostatic biopsy; all preoperative investigations were negative for prostate cancer.

Table 1.
Baseline characteristics of patients with incidental prostate cancer (iPCa).

Age, years	**73 (48–90)**
Comorbidities	
Arterial hypertension	34 (82.9%)
Diabetes mellitus	20 (48.8%)
Dyslipidemia	32 (78.0%)
Renal transplant recipient	4 (9.8%)
Current smoker	3 (7.3%)
Antiplatelet therapy	13 (31.7%)
Anticoagulant therapy	4 (9.8%)
BPH medical therapy	
None	7 (17.1%)
α-blocker	10 (24.4%)
5-alpha-reductase inhibitor (5-ARI)	2 (4.9%)
α-blocker + 5-ARI	17 (41.5%)
Serenoa repens	1 (2.4%)
Serenoa repens + α-blocker	3 (7.3%)
Serenoa repens + α-blocker + 5-ARI	1 (2.4%)
Preoperative tests	
PSA, ng/mL	3.06 (0.10–27.90)
Prostate volume, cm³	45 (20–197)
PSA density, ng/mL/cm³	0.064 (0.003–0.636)
Prostate mpMRI performed	2 (4.9%)
Preoperative prostate biopsy	3 (7.3%)

Surgical outcomes and perioperative data

As summarized in Table 2, 32/41 (78%) patients underwent TURP and 9/41 (22%) underwent simple prostatec-

Table 2.
Surgical and perioperative variables in patients with iPCa.

Type of surgery	
Transurethral resection of the prostate (TURP)	32 (78.0%)
Simple prostatectomy	9 (22.0%)
Surgical indication	
Refractory lower urinary tract symptoms (LUTS)	17 (41.5%)
Chronic urinary retention with indwelling catheter	16 (39.0%)
Persistently elevated post-void residual urine	8 (19.5%)
Length of hospital stay, days	2.5 (1–9)
Bladder catheter at discharge	38 (92.7%)
Postoperative catheterization, days	7 (4–20)
Emergency department visits within 30 days	8 (19.5%)

tomy. Surgical indications were refractory *lower urinary tract symptoms* (LUTS) in 17 (41.5%), chronic indwelling catheter due to urinary retention in 16 (39.0%), and persistently elevated post-void residuals in 8 (19.5%).

The median length of stay was 2.5 days (range, 1-9). Preoperative haemoglobin was 14.5 g/dL (mean); 1 patient (2.4%) required postoperative red-blood-cell transfusion due to pre-existing chronic anaemia (pre-op Hb 9 g/dL) (Clavien-Dindo II). Other postoperative events included hematuria requiring continuous bladder irrigation (managed conservatively; Clavien I-II), one febrile lower urinary tract infection (Clavien II), and one transient left lower-limb weakness that resolved with conservative measures (Clavien I).

At discharge, 38/41 (92.7%) patients left with an indwelling urethral catheter; the median catheterization time was 7 days (range, 4-20). Within 30 days, 8/41 (19.5%) required emergency department evaluation, predominantly for recurrent hematuria. During early postoperative follow-up, acute urinary retention occurred in 4 patients (9.8%); 3 evolved to chronic indwelling catheterization due to acontractile bladder, confirmed on urodynamic testing.

Incidental cancer diagnosis and pathological features

On histopathological assessment (Table 3), the resected tissue weight had a median of 13.5 g (range, 3-107 g). Tumor involvement represented a median of 10% of the specimen (range, 1-90%). According to the ISUP Grade Group system, ISUP 1 was the most frequent (20/41; 48.8%), followed by ISUP 2 (11/41; 26.8%); ISUP 3 and ISUP 4 (3/41; 7.3%), and ISUP 5 (4/41; 9.8%), indicating a predominance of low-grade disease but with a relevant subset of higher-grade tumors.

Preoperative PSA and PSA density were compared between low-grade (ISUP 1; n = 20) and high-grade (ISUP ≥ 4; n = 7) cancers using the Mann-Whitney U test (Table 3).

The postoperative PSA showed a marked reduction compared with preoperative values, with a mean of 0.95 ng/mL (range, 0.04-27.6 ng/mL).

Following the postoperative diagnosis of iPCa, multiparametric MRI (mpMRI) was performed in 14/41 (34.1%) patients based on clinical risk stratification. PI-RADS ≥ 4 lesions were detected in 5/14, while 9/14 had PI-RADS ≤ 3. All 14 underwent transrectal biopsy: patients with PI-RADS ≤ 3 had systematic cores, and those with PI-RADS ≥ 4 received systematic plus targeted cores. Biopsy results were negative in 7 patients, ISUP 1 in 4, and ISUP ≥ 2 in 3.

Predictive factors of iPCa

Among the 735 men operated, 41 (5.6%) had iPCa on final histopathology and 694 (94.4%) had no carcinoma. Baseline age was comparable between groups (p = 0.184). In contrast, PSA and *PSA density* (PSAD) were higher and prostate volume was lower in the iPCa group (all p < 0.001), as shown in Table 5.

Figure 1.
Flow diagram of patient selection for the iPCa cohort (November 2020-December 2024).

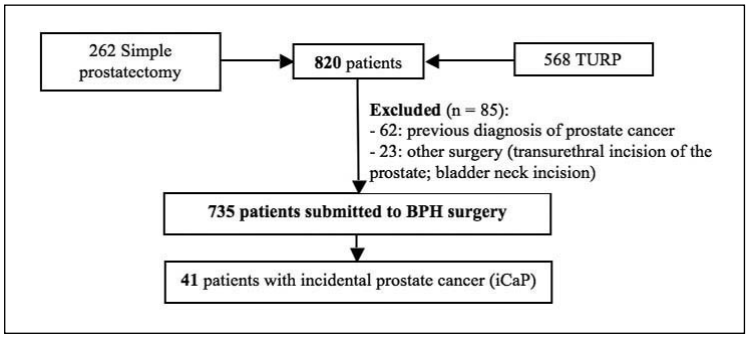

Table 3.
Post-diagnosis histopathology and imaging work-up in patients with iPCa.

Age, years	**73 (48–90)**
Surgical specimen	
Resected tissue weight, g	13.5 (3-107)
Tumour involvement, % of specimen	10 (1-90)
ISUP Grade Group	
ISUP 1	20 (48.8%)
ISUP 2	11 (26.8%)
ISUP 3	3 (7.3%)
ISUP 4	3 (7.3%)
ISUP 5	4 (9.8%)
Post-diagnosis mpMRI performed	14/41 (34.1%)
PI-RADS ≤ 3	9/14 (64.3%)
PI-RADS ≥ 4	5/14 (35.7%)
Post-diagnosis biopsy performed	14/41 (34.1%)
Negative	7/14 (50.0%)
ISUP 1	4/14 (28.6%)
ISUP ≥ 2	3/14 (21.4%)

PI-RADS: Prostate Imaging Reporting and Data System.
ISUP: International Society of Urological Pathology.

Table 4.
Comparison of preoperative PSA and PSA density between ISUP Grade Group 1 and ≥ 4.

	ISUP Grade 1 (n = 20)	ISUP Grade ≥ 4 (n = 7)	p-value
Preoperative PSA (ng/mL)	1.97 (1.40–3.20)	7.80 (6.50–11.00)	< 0.001
PSA density (ng/mL/cm³)	0.054 (0.042–0.132)	0.220 (0.140–0.350)	< 0.001

Table 5.
Comparative analysis between patients with and without iPCa: Values shown as median and interquartile range [IQR]; p-values from Mann-Whitney U test.

	BPH (n = 694)	iPCa (n = 41)	p-value
Age, years	70 (66-76)	72 (67-78)	0.184
PSA, ng/mL	1.9 (1.2-2.8)	4.8 (3.3-6.9)	< 0.001
Prostate volume, mL	66 (50-80)	47 (35-60)	< 0.001
PSA density, ng/mL/cm³	0.029 (0.020-0.040)	0.099 (0.070-0.140)	< 0.001

Table 6.
Multivariable logistic regression model.

	Odds ratio (95% CI)	p-value
Age (per year)	1.03 (0.98-1.09)	0.204
PSAD (per 0.01 ng/mL/cm³)	1.28 (1.14-1.47)	< 0.001

In multivariable logistic regression including age and PSAD, only PSAD remained independently associated with iPCa (per 0.01 ng/mL/cm³: OR 1.28, 95% CI 1.14-1.47, p < 0.001), whereas age was not significant (per year: OR 1.03, 95% CI 0.98-1.09, p = 0.204) (Table 6).
Diagnostic accuracy analyses confirmed the superiority of PSAD over PSA. The AUC for PSAD was 0.86 (95% CI 0.79-0.91) versus 0.80 (95% CI 0.73-0.87) for PSA (Figure 2).
The Youden-optimized cut-off for PSAD (≈ 0.15 ng/mL/cm³) yielded sensitivity 0.82, specificity 0.78, PPV 0.39, and NPV 0.96; for PSA (4.3 ng/mL), sensitivity and specificity were 0.75 and 0.73 with PPV 0.32 and NPV 0.95 (Table 7). Collectively, these results indicate that PSAD is the strongest preoperative marker for iPCa in this cohort.

Figure 2.
Left: ROC curve for preoperative PSA; Right: ROC curve for PSA density.

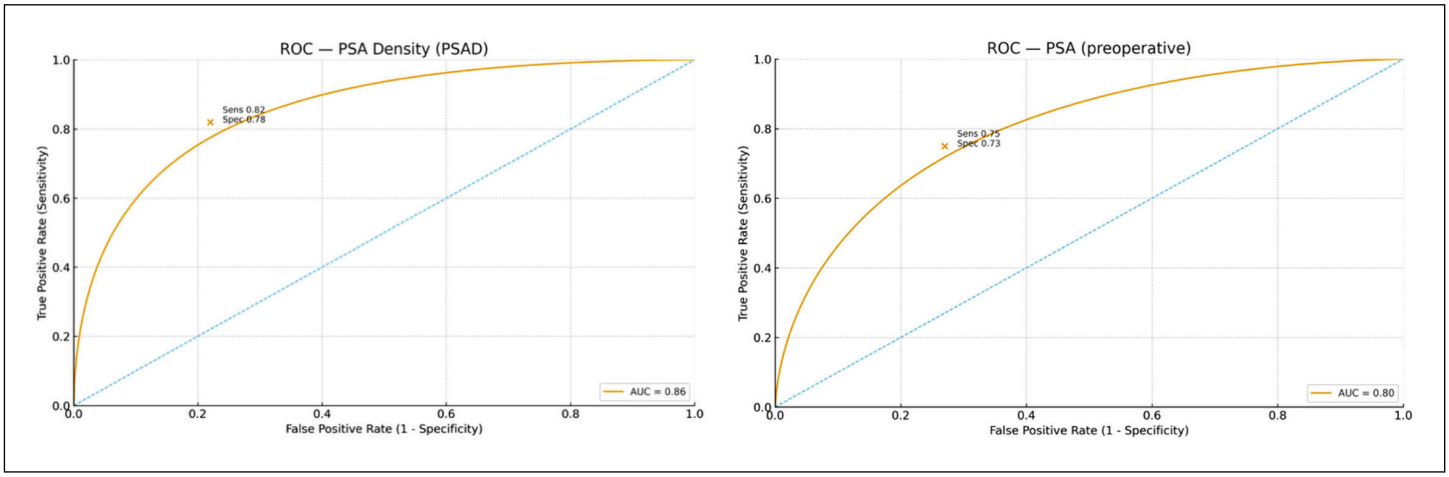

Table 7.
Diagnostic performance of PSA and PSAD for iPCa prediction.

	Optimal cut-off (Youden)	Sensitivity	Specificity	PPV	NPV	AUC (95% CI)
PSA, ng/mL	4.3	0.75	0.73	0.32	0.95	0.80 (0.73-0.87)
PSA density ng/mL/cm³	0.15	0.82	0.78	0.39	0.96	0.86 (0.79-0.91)

PPV: positive predictive value; NPV: negative predictive value.

Oncological follow-up

The median follow-up was 24 months (range 10-50). At last follow-up, six patients had died – four from non-prostate cancer causes and two from disease progression. Among those managed with active surveillance (n = 16), the median age at diagnosis was 68 years (IQR 62.8-70.0; range 48-73), and the most recent PSA showed a median of 1.12 ng/mL (IQR 0.64-1.87; range 0.25-4.39); one patient (6.3%) requiring re-evaluation for PSA rise, was upgraded to ISUP Grade Group 2 on repeat biopsy, and received definitive radiotherapy. In the watchful waiting cohort (n = 18), the median age was 76.5 years (IQR 73.5-79.0; mean 77.1 ± 5.9); 3 patients (16.7%) died of non-urological causes; among 15 survivors with recent laboratory data, the latest PSA had a median of 1.50 ng/mL (IQR 0.77-2.23; range 0.15-6.35).
In patients treated with hormonal therapy (n = 7), the median age at diagnosis was 80.0 years (IQR 77.5-85.5; mean 80.9 ± 5.7); two (28.6%) died from progressive disease, and among the remaining five with recent tests, the latest PSA showed a median of 0.45 ng/mL (IQR 0.22-0.50; range 0.06-6.50). At last contact, androgen-deprivation regimens included bicalutamide monotherapy in 1/7 (14.3%), LHRH agonist monotherapy in 3/7 (42.9%), and LHRH agonist plus an androgen-receptor pathway inhibitor in 3/7 (42.9%).

Discussion

In our cohort, the mean age at diagnosis of iPCa was 73 years, reflecting a predominantly elderly population. This is consistent with the epidemiology of both BPH and prostate cancer aligning with previous TURP studies, including those published by *Porcaro et al.* and *Otto et al.*, in which the mean patient age ranged from 70 to 75 years (6, 8). The high prevalence of metabolic comorbidities observed in our patients reflects the typical BPH surgical population described in previous studies and may influence both perioperative management and long-term oncological management and outcomes (8).
In this series prostate cancer iPCa was found in 5.6% of

BPH surgeries – toward the lower end of contemporary series and below pooled estimates around 8-10%. The figure is consistent with reports from the PSA era showing declining incidental detection in modern BPH surgery cohorts (TURP/HoLEP) (2, 5-7) and with meta-analyses highlighting center-level variability driven by preoperative biopsy and imaging practices (1, 4).

The mean preoperative PSA in our study population was 3.05 ng/mL, a value not typically considered suspicious for prostate cancer. In their meta-analysis, *Guo et al.* identified elevated PSA as the strongest independent predictor of iPCa risk, although no specific PSA threshold indicative of malignancy was defined (1). *Wang et al.* also confirmed the association between elevated preoperative PSA and incidental cancer diagnosis (4). The mean prostatic volume, measured by ultrasound, was 45 cm^3, comparable to other TURP cohorts and in agreement with the inverse relationship between prostatic tissue weight and cancer detection described by *Wang et al.* (4).

Pathological analysis revealed that almost half of our tumors (48.8%) were ISUP Grade Group 1. This distribution is in line with the 73% reported by *Porcaro et al.* (8) and the predominance of low-grade disease (60-75%) highlighted in the meta-analysis by *Guo et al.* (1). The presence of ISUP ≥ 2 lesions in our series, including four ISUP 5 tumors, underscores that a relevant proportion of iPCa is clinically significant. *Otto et al.* found higher-grade disease in approximately 20% of incidental cases (6), and *Abedi et al.* emphasized that not all iPCa can be managed conservatively (9).

Preoperative determinants were concordant with higher-level evidence. Patients with iPCa had higher PSA/PSA PSAD and smaller prostates, whereas age was not discriminatory; in multivariable analysis PSAD remained the strongest independent predictor (AUC 0.86). Meta-analyses and contemporary reviews likewise identify PSAD as the most informative marker for iPCa in BPH surgery candidates, with an inverse association between gland size and cancer detection; thresholds near 0.15 ng/mL/cm^3 provide a pragmatic balance between sensitivity and specificity (1, 4, 10, 11). Taken together, our findings support integrating PSAD into preoperative pathways to guide selective mpMRI and/or biopsy before benign prostatic surgery, particularly in borderline cases.

Oncological follow-up in this cohort was largely reassuring over a median of 24 months. Most patients were managed conservatively – active surveillance or watchful waiting – with stable PSA profiles: in active surveillance, only 1/16 (6.3%) triggered re-evaluation for PSA rise, resulting in upgrade to ISUP Grade Group 2, and received definitive radiotherapy; in watchful waiting, the latest PSA among survivors remained low (median 1.50 ng/mL). Overall mortality was dominated by non-prostate cancer causes (4/6 deaths), consistent with the advanced age and comorbidity of the watchful-waiting subgroup and aligns with recommendations to prioritize conservative strategies for pT1a and low-risk pT1b disease while reserving definitive treatment for pathological upgrade or biochemical progression (9, 10, 13).

A smaller subset required systemic therapy upfront, reflecting biologically aggressive presentations that can still be detected incidentally. Among those on hormonal therapy (n = 7), two cancer-related deaths occurred despite contemporary regimens (including LHRH agonist plus androgen receptor pathway inhibitors in 3/7), underscoring that incidental detection does not preclude clinically significant disease. This observation mirrors reports that 18-25% of iPCa may be clinically relevant (1, 6) and supports a risk-adapted follow-up anchored in postoperative PSA kinetics (with 5-ARI adjustment when applicable), PSA density, and selective mpMRI to refine stratification (4, 10). Taken together – and in line with population-level comparisons showing convergence of outcomes by stage/grade rather than route of diagnosis (13, 15) – our data reinforce conservative management as the default for low-risk iPCa, with vigilant triggers for escalation in the minority exhibiting adverse biology.

Limitations and future perspectives

This study has inherent limitations, including its retrospective and single-center design, which may introduce selection bias. The lack of systematic postoperative multiparametric MRI or biopsy hinders precise evaluation of residual disease. Furthermore, the follow-up duration may not fully reflect long-term oncological outcomes, particularly in conservatively managed patients.

Future research should focus on prospective multicenter studies with standardized postoperative imaging or biopsy protocols to better characterize residual disease. Longer follow-up periods are needed to assess long-term outcomes and progression risk, especially for ISUP ≥ 2 cases. Incorporating PSA density, advanced biomarkers, and functional outcomes may also help refine risk stratification and management strategies for iPCa.

DECLARATIONS

Ethical approval and consent for participate: *Ethical approval was not required for this retrospective study based on anonymized clinical data, in accordance with institutional and national research ethics guidelines.*

Availability of data and material: *The datasets generated and/or analyzed during the current study are not publicly available due to institutional restriction, but they are available from the corresponding author upon reasonable request.*

Competing interests: *The authors declare that they have no competing interests.*

Funding: *This study received no financial support.*

Authors' contributions: *Rui Pedrosa, João Lorigo, Bárbara Figueiredo, and Ana João Guerra contributed to the conceptualization and investigation of the study. Vasco Quaresma, Miguel Eliseu, and Arnaldo Figueiredo provided methodological support. Formal data analysis was performed by Rui Pedrosa, João Lorigo, Ana João Guerra, and Vasco Quaresma. The original draft was prepared by Rui Pedrosa, Vasco Quaresma, Miguel Eliseu, and Paulo Temido, while all authors contributed to the review and editing of the manuscript. Arnaldo Figueiredo supervised the study. All authors critically revised the work for important intellectual content and approved the final submitted version.*

Conclusions

In our study, the incidence of iPCa was comparable to rates reported in other contemporary studies. Almost half of cases representing low-grade and potentially indolent disease. Nevertheless, the presence of clinically significant prostate cancer (ISUP ≥ 2), underscores the need for careful pathological assessment and structured postoperative management. Preoperative PSA density appeared as relevant predictor of incidental cancer, reinforcing their role in risk stratification. Future prospective studies incorporating multiparametric MRI and other biomarkers are warranted to refine the management of this group of patients.

References

1. Guo Z, He J, Huang L, 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; 48:915-929.

2. Capogrosso P, Capitanio U, Vertosick EA, et al. Temporal trend in incidental prostate cancer detection at surgery for benign prostatic hyperplasia. Urology. 2018; 122:152-57.

3. Gratzke C, Bachmann A, Descazeaud A, et al. EAU Guidelines on the Assessment of Non-neurogenic Male Lower Urinary Tract Symptoms including Benign Prostatic Obstruction. Eur Urol. 2015; 67:1099-1109.

4. Wang Y, Li X, Yang H, et al. 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.

5. Yilmaz M, Toprak T, Suarez-Ibarrola R, et al. Incidental prostate cancer after holmium laser enucleation of the prostate–A narrative review. Andrologia. 2022; 54:e14332.

6. Otto B, Barbieri C, Lee R, et al. Incidental prostate cancer in transurethral resection of the prostate specimens in the modern era. Adv Urol. 2014; 2014:627290.

7. Shvero A, Teplitsky S, Kloniecke E, Das AK. Incidental prostate cancer at holmium laser enucleation of the prostate. Can J Urol. 2021; 28:10900-10906.

8. Porcaro AB, Tafuri A, Inverardi D, et al. Incidental prostate cancer after transurethral resection of the prostate: analysis of incidence and risk factors in 458 patients. Minerva Urol Nephrol. 2021; 73:471-480.

9. Abedi AR, Ghiasy S, Fallah-karkan M, et al. The management of patients diagnosed with incidental prostate cancer: narrative review. Res Rep Urol. 2020; 12:105-109.

10. Li X, Chen Y, Zhang Q, et al. Modern predictors and management of incidental prostate cancer (iPCa). Prostate. 2024; 84:1061-1073.

11. Anract J, Roussel E, Lefèvre L, et al. Incidental prostate cancer in BPH surgery: contemporary predictors and clinical implications. Curr Probl Cancer. 2025; 49:100193.

12. Leni R, Dinh A, Durand X, et al. Management and oncologic outcomes of incidental prostate cancer detected after surgery for BPH. J Urol. 2024; 212:445-454.

13. Leni R, Lefebvre D, Hsu C, et al. Oncologic outcomes of incidental versus biopsy-detected prostate cancer: a comparative study. Prostate Cancer Prostatic Dis. 2024; 27:981-990.

14. Di Bello C, Wessells H, Mallick R, et al. Cancer-specific mortality after radical prostatectomy versus radiotherapy in incidental prostate cancer: a population-based analysis. Urol Oncol. 2024; 42:1073.e1-1073.e9.

15. Bologna G, Sanchez M, Patel V, et al. Incidental prostate cancer in patients treated for BPH: a contemporary population-based analysis. Diagnostics (Basel). 2024; 14:677.

Correspondence

Rui Pedrosa
rui.mdp93@gmail.com

João Lorigo
joaolorigo@gmail.com

Ana João Guerra
anajoaoguerra@gmail.com

Bárbara Figueiredo
barbarajrf@icloud.com

Vasco Quaresma
vpdquaresma@gmail.com

Miguel Eliseu
mglnobre@gmail.com

Paulo Temido
ptemido@gmail.com

Arnaldo Figueiredo
ajcfigueiredo@gmail.com

Department of Urology and Renal Transplantation, ULS Coimbra, Coimbra, Portugal
Praceta Professor Mota Pinto, Celas, 3004-561 Coimbra, Portugal