Evaluation of Predictive Factors for Prostate Cancer Detection in PI-RADS 2 Lesions

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Purpose: To evaluate clinical, biochemical, and hematological parameters as predictors of prostate cancer (PCa) in patients with Prostate Imaging Reporting and Data System (PI-RADS) 2 lesions.

Materials and Methods: A total of 955 patients underwent multiparametric magnetic resonance imaging (mpMRI) during the study period, of whom 268 had PI-RADS 2 lesions. Among these, 147 patients with histopathological diagnoses, including 122 benign and 25 malignant cases, were included. Clinical, biochemical, and hematological parameters were compared between groups using appropriate statistical tests. Receiver operating characteristic (ROC) curve analyses were performed to evaluate diagnostic performance.

Results: Prostate-specific antigen (PSA) density (P = .044), neutrophil count (P = .019), monocyte count (P = .021), neutrophil-to-platelet ratio (NPR; P = .016), and monocyte-to-platelet ratio (MPR; P = .019) differed significantly between the malignant and benign groups. Compared with the benign group, neutrophil count, monocyte count, NPR, and MPR were lower, whereas PSA density was higher in patients with PCa. ROC analyses demonstrated moderate discriminatory performance for PSA density (area under the curve [AUC], 0.654; 95% confidence interval [CI], 0.561–0.739; P = .007), monocyte count (AUC, 0.655; 95% CI, 0.546–0.754; P = .011), MPR (AUC, 0.657; 95% CI, 0.547–0.755; P = .037), neutrophil count (AUC, 0.635; 95% CI, 0.523–0.737; P = .046), and NPR (AUC, 0.644; 95% CI, 0.536–0.743; P = .055).

Conclusion: PSA density demonstrated moderate discriminatory performance for detecting PCa in biopsied patients with PI-RADS 2 lesions. Although several hematological indices also showed moderate discriminatory performance, their clinical utility remains limited. Larger prospective studies are required to validate these findings before clinical implementation.

Keywords: prostatic neoplasms; magnetic resonance imaging; prostate-specific antigen; biomarkers, tumor; hematologic tests; predictive value of testst

INTRODUCTION

Prostate cancer (PCa) remains one of the most common malignancies in men worldwide, and despite improvements in imaging and clinical markers, accurate early detection remains a diagnostic challenge.[1] Accurate diagnosis has become increasingly important in the era of personalized PCa management, as expanding therapeutic options have further emphasized the need for accurate risk stratification at the time of diagnosis.[2,3] Multiparametric magnetic resonance imaging (mpMRI) has emerged as a powerful imaging tool in PCa evaluation. It has been standardized by the Prostate Imaging Reporting and Data System (PI-RADS) version 2.1, which allows risk stratification based on lesion characteristics.[4]

Although PI-RADS 2 lesions are typically considered to have a low likelihood of clinically significant prostate cancer (csPCa), and current guidelines generally do not recommend routine biopsy in the absence of additional clinical risk factors, meta-analytic evidence indicates that approximately 5% to 10% of patients with PI-RADS 2 findings may still harbor PCa.[5-7] Nevertheless, a subset of patients may still elect to undergo prostate biopsy after being informed of this residual risk through a shared decision-making process. Findings from a large high-volume center by Jahnen et al. further support the need for careful risk assessment, demonstrating that csPCa may still be detected in lesions considered to be at low risk.[8] Current European Association of Urology (EAU) Guidelines recommend that prostate biopsy decisions should not rely solely on mpMRI findings but should also incorporate additional clinical risk factors, including prostate-specific antigen (PSA) density, digital rectal examination findings, and individual patient characteristics.[6]

To enhance risk stratification in PI-RADS 2 lesions, adjunctive predictors are increasingly being explored.[9] PSA density, computed as serum PSA divided by prostate volume, has shown promise in improving diagnostic accuracy when combined with PI-RADS scoring.[10] Additionally, inflammatory markers derived from complete blood count, such as neutrophil, lymphocyte, and monocyte counts and their ratios, are emerging as cost-effective, noninvasive indicators of tumor presence and behavior.[11]

However, few studies have specifically investigated predictive markers exclusively in patients with PI-RADS 2 lesions, as most previous studies have evaluated broad-

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Table 1. Data are presented as mean ± SD or median (IQR), as appropriate. Comparisons were performed using the independent-samples t-test or Mann–Whitney *U* test.

Variables	Benign (n = 122)	Malignant (n = 25)	*P* value
Age (years)	66.24 ± 5.75	66.72 ± 9.38	.740
PSA (ng/mL)	5.35 (4.01-8.94)	5.72 (4.60-8.00)	.630
Prostate volume (mL)	67.00 (53.00-107.00)	53.00 (43.00-73.50)	.086
PSA density (ng/mL/mL)	0.08 (0.05-0.13)	0.09 (0.07-0.14)	.044
Albumin (g/dL)	4.33 ± 0.36	4.47 ± 0.37	.147
Neutrophil count (109/L)	5.73 (4.06-7.87)	4.10 (3.40-5.35)	.019
Lymphocyte count (×109/L)	1.99 (1.50-2.34)	1.60 (1.35-2.24)	.279
Monocyte count (109/L)	0.53 (0.37-0.70)	0.42 (0.34-0.47)	.021
Platelet count (109/L)	249.48 ± 54.86	254.90 ± 76.55	.719
NLR	2.84 (1.95-4.20)	2.18 (1.84-3.02)	.170
NPR	0.021 (0.018-0.029)	0.017 (0.013-0.020)	.016
NMR	11.97 (8.98-15.91)	10.83 (7.83-14.13)	.401
LPR	0.078 (0.061-0.109)	0.066 (0.054-0.082)	.151
LMR	4.04 (3.08-6.54)	4.68 (3.69-5.66)	.634
MPR	0.020 (0.017-0.028)	0.015 (0.012-0.023)	.019
PNI	50.10 (41.15-55.12)	54.20 (45.00-56.20)	.375

Abbreviations: PSA, prostate-specific antigen; NLR, neutrophil-to-lymphocyte ratio; NPR, neutrophil-to-platelet ratio; NMR, neutrophil-to-monocyte ratio; LPR, lymphocyte-to-platelet ratio; LMR, lymphocyte-to-monocyte ratio; MPR, monocyte-to-platelet ratio; PNI, prognostic nutritional index.

er cohorts of patients with negative mpMRI findings or higher PI-RADS categories.[12,13] Therefore, the present study aimed to evaluate the diagnostic performance of PSA density and selected hematological parameters for predicting PCa in patients with PI-RADS 2 lesions. By identifying reliable markers, we sought to improve risk stratification and support biopsy decision-making in this challenging group of patients.

MATERIALS AND METHODS

Patient Selection

Following approval from the local ethics committee (Decision No. 407 - 407 - 05; date: July 10, 2024), this retrospective study was conducted using patient records from our institutional database, covering the period from January 2021 to May 2024. The requirement for written informed consent was waived by the ethics committee because of the retrospective nature of the study. All patient data were anonymized before analysis, and confidentiality was strictly maintained in accordance with the Declaration of Helsinki. Patients who underwent mpMRI followed by prostate biopsy and were identified as having PI-RADS 2 lesions were eligible for inclusion. Exclusion criteria were a prior history of PCa, previous prostate surgery, prior androgen-deprivation therapy, incomplete clinical data, or inadequate biopsy specimens. All demographic, clinical, laboratory, imaging, and pathological data were retrospectively collected from the institutional electronic medical record system.

At our institution, patients with PI-RADS 2 lesions are routinely informed that, despite the low likelihood of csPCa, malignancy may still be detected in a small proportion of cases. After counseling regarding the potential benefits and risks of prostate biopsy, the decision to undergo biopsy is made through shared decision-making between the patient and the treating urologist. Patients who underwent biopsy and had available histopathological results were included in the present study, whereas patients who declined biopsy continued clinical follow-up and were not included in the analysis. Patients with active urinary tract infection or acute prostatitis at the time of the biopsy decision received appropriate medical treatment, and prostate biopsy was performed only after resolution of the infection. Laboratory parameters included in the study were obtained after resolution of the inflammatory process.

Clinical and Laboratory Data

Demographic characteristics, prostate volume, serum PSA levels, serum albumin levels, and complete blood count parameters were collected from the institutional electronic medical record system. PSA density was calculated as serum PSA (ng/mL) divided by prostate volume (mL). Hematological parameters included neutrophil, lymphocyte, monocyte, and platelet counts, as well as derived indices such as the neutrophil-to-lymphocyte ratio (NLR), neutrophil-to-monocyte ratio (NMR), neutrophil-to-platelet ratio (NPR), lymphocyte-to-monocyte ratio (LMR), lymphocyte-to-platelet ratio (LPR), monocyte-to-platelet ratio (MPR), platelet-to-lymphocyte ratio (PLR), and prognostic nutritional index (PNI). The derived hematological indices were calculated as follows: NLR = neutrophil count /

Table 2. ROC analyses of significant variables associated with prostate cancer detection in PI-RADS 2 lesions. Optimal cut-off values were calculated using the Youden index. Sensitivity, specificity, PPV, and NPV are presented with their corresponding 95% CIs, where applicable.

Variable	AUC (95% CI)	P value	Cut-off	PPV (95% CI)	NPV (95% CI)	Sensitivity (95% CI)	Specificity (95% CI)
PSA density (ng/mL/mL)	0.654 (0.561–0.739)	.007	0.0617	24.4 (21.0–28.0)	97.6 (85.4–99.6)	95.0 (75.1–99.9)	40.4 (30.7–50.7)
Neutrophil count (109/L)	0.635 (0.523–0.737)	.045	5.68	31.5 (26.1–37.4)	93.5 (79.2–98.2)	89.5 (66.9–98.7)	43.9 (31.7–56.7)
Monocyte count (109/L)	0.655 (0.546–0.754)	.011	0.51	32.1 (27.6–38.3)	96.8 (81.9–99.5)	94.7 (74.0–99.9)	45.6 (33.5–58.1)
NPR	0.644 (0.536–0.743)	.055	0.0205	31.9 (24.7–40.1)	88.1 (77.1–94.2)	75.0 (50.9–91.3)	53.6 (41.2–65.7)
MPR	0.657 (0.547–0.755)	.037	0.00179	37.1 (27.9–49.7)	88.7 (79.9–93.9)	68.4 (43.4–87.4)	67.7 (55.7–78.8)

Abbreviations: ROC, receiver operating characteristic; AUC, area under the curve; CI, confidence interval; PPV, positive predictive value; NPV, negative predictive value; PSA, prostate-specific antigen; NPR, neutrophil-to-platelet ratio; MPR, monocyte-to-platelet ratio.

Note: AUC interpretation: < 0.60, poor discrimination; 0.60–0.69, moderate discrimination; 0.70–0.79, acceptable discrimination; 0.80–0.89, excellent discrimination; ≥ 0.90, outstanding discrimination.

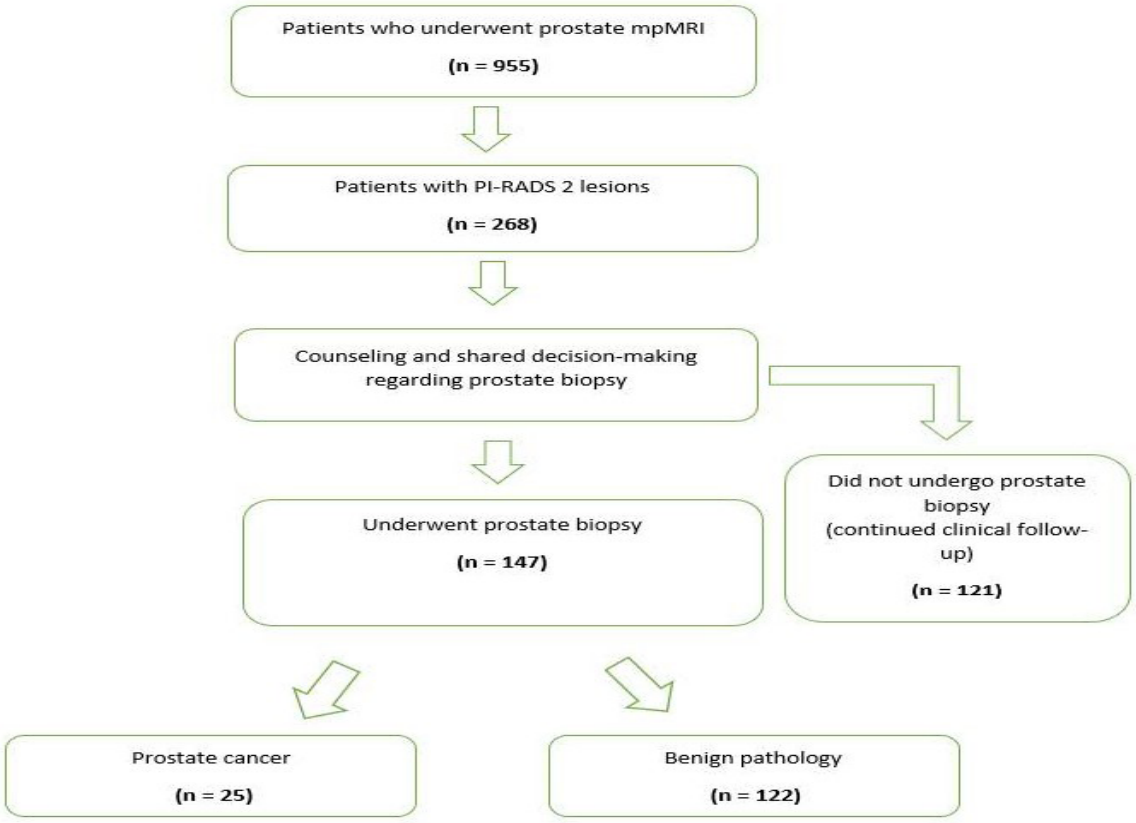

Figure 1. Flow diagram showing patient selection and inclusion process.

lymphocyte count, NMR = neutrophil count / monocyte count, NPR = neutrophil count / platelet count, LMR = lymphocyte count / monocyte count, LPR = lymphocyte count / platelet count, MPR = monocyte count / platelet count, PLR = platelet count / lymphocyte count, and PNI = serum albumin (g/L) + 5 × total lymphocyte count ($\times 10^9$/L).

Imaging and Biopsy Protocol

All patients underwent mpMRI using a 3.0-T MRI scanner before prostate biopsy. The imaging protocol included axial, sagittal, and coronal T2-weighted imaging, diffusion-weighted imaging (DWI) with high b values, apparent diffusion coefficient (ADC) maps, and dynamic contrast-enhanced (DCE) imaging according to the institutional protocol. Prostate lesions were evaluated according to PI-RADS version 2.1 by radiologists at our institution. Prostate volume was measured on MRI using the ellipsoid formula and was used to calculate PSA density.

Subsequently, all included patients underwent systematic 12-core transrectal ultrasound-guided prostate biopsy. MRI-targeted biopsy was not performed because none of the lesions was categorized as PI-RADS $\geq$ 3. Histopathological evaluation was performed according to the 2014 International Society of Urological Pathology (ISUP) Gleason grading system. csPCa was defined as ISUP grade group $\geq$ 2 (Gleason score $\geq$ 3 + 4).

Statistical Analyses

Descriptive statistics were expressed as mean ± standard deviation (SD) or median (interquartile range [IQR]), as appropriate, according to the distribution of the data. Data distribution was assessed using the Shapiro–Wilk test, and homogeneity of variances was evaluated using Levene's test. Between-group comparisons were performed using Student's t-test or the Mann–Whitney *U* test, as appropriate. Categorical variables were analyzed using the Pearson chi-square test when the assumptions for the test were met; otherwise, Fisher's exact test was applied when expected cell counts were less than 5. Receiver operating characteristic (ROC) curve analyses were performed for variables that showed statistically significant differences between the benign and malignant groups. The area under the curve (AUC) with 95% confidence intervals (CIs) was calculated. Optimal cut-off values were determined using Youden's index. Sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) corresponding to the selected cut-off values were obtained from the ROC analysis. A two-sided *P* value < .05 was considered statistically significant. Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 23.0 (IBM Corp., Armonk, NY, USA), and MedCalc Statistical Software version 23.6.2 (MedCalc Software Ltd., Ostend, Belgium).

RESULTS

Demographic and Clinicopathological Characteristics

A total of 955 patients underwent prostate mpMRI

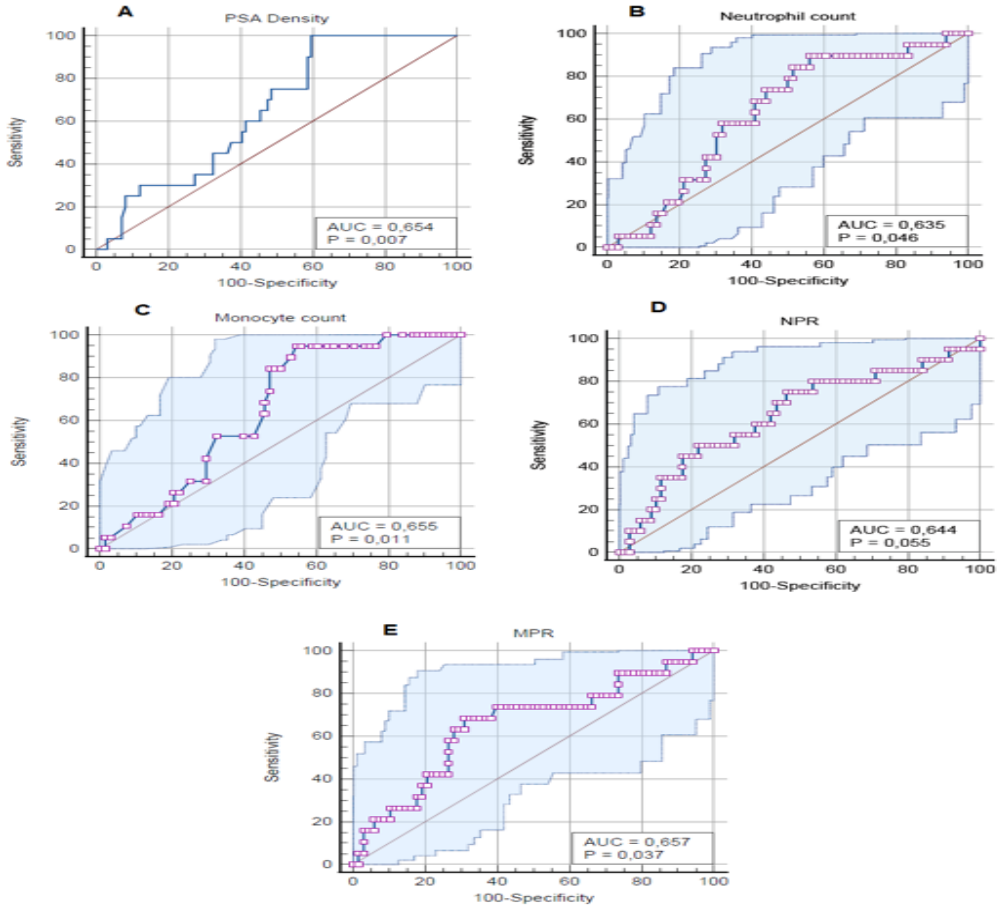

Figure 2. Receiver operating characteristic (ROC) curves for (A) prostate-specific antigen (PSA) density, (B) neutrophil count, (C) monocyte count, (D) neutrophil-to-platelet ratio (NPR), and (E) monocyte-to-platelet ratio (MPR).
AUC values and 95% confidence intervals are provided for each marker.
These curves illustrate the discriminatory performance of each biomarker in differentiating benign from malignant cases.

during the study period. Of these, 268 were reported to have PI-RADS 2 lesions. Among these patients, 147 underwent prostate biopsy, had histopathological diagnoses, and were included in the final analysis: 122 (83%) had benign pathology, and 25 (17%) had PCa. The study flow is summarized in **Figure 1**. The mean age was comparable between the benign and malignant groups (benign: 66.24 ± 5.75 years; malignant: 66.72 ± 9.38 years), with no statistically significant difference (P = .740). Within the malignant subgroup (n = 25), 19 patients (76%) had a Gleason score of 3 + 3 (ISUP grade group 1), 4 (16%) had a Gleason score of 3 + 4 (ISUP grade group 2), 1 (4%) had a Gleason score of 4 + 3 (ISUP grade group 3), and 1 (4%) had a Gleason score of 4 + 4 (ISUP grade group 4). According to the ISUP grading system, csPCa was identified in 6 of 25 patients with PCa (24.0%), corresponding to 6 of 147 biopsied patients (4.1%).

Hematologic and Biochemical Indices Between Benign and Malignant Groups

Among the 147 patients with PI-RADS 2 lesions, 122 (83%) had benign pathology, whereas 25 (17%) were diagnosed with PCa. PSA density (P = .044), neutrophil count (P = .019), monocyte count (P = .021), NPR (P = .016), and MPR (P = .019) differed significantly between the benign and malignant groups. Compared with the benign group, neutrophil count, monocyte count, NPR, and MPR were lower in the malignant group, whereas PSA density was higher. In contrast, age (P = .740), platelet count (P = .719), albumin level (P = .147), and other hematological parameters and indices showed no significant differences between the groups. Detailed results are presented in **Table 1**.

ROC Curve Analyses

ROC curve analyses were performed for variables that showed significant differences between the benign and malignant groups. Among the evaluated variables, PSA density demonstrated low-to-moderate discriminatory performance for predicting PCa in patients with PI-RADS 2 lesions (AUC, 0.654; 95% CI, 0.561–0.739; P = .007). Other hematological markers, including neutrophil count (AUC, 0.635; 95% CI, 0.523–0.737; P = .046), monocyte count (AUC, 0.655; 95% CI, 0.546–0.754; P = .011), NPR (AUC, 0.644; 95% CI, 0.536–0.743; P = .055), and MPR (AUC, 0.657; 95% CI, 0.547–0.755; P = .037), also showed low-to-moder-

ate discriminatory power.
Optimal cut-off values were calculated using the Youden index. PSA density at a threshold of 0.06 yielded a sensitivity of 95.0% and a specificity of 40.4%. In contrast, the hematological indices demonstrated moderate sensitivity and relatively low specificity. The ROC curves are illustrated in Figure 2, and detailed diagnostic performance measures, including sensitivity, specificity, PPV, and NPV with their 95% CIs, are presented in **Table 2**.

DISCUSSION

In this study, we aimed to investigate whether PSA density and selected hematologic parameters could enhance the prediction of PCa in patients with PI-RADS 2 lesions. Although these lesions are typically considered to pose a low risk for csPCa, mounting evidence suggests that a meaningful subset, estimated at approximately 5%, may indeed harbor cancer.[14] This uncertainty complicates biopsy decision-making in clinical practice, raising the question of whether every PI-RADS 2 lesion warrants invasive sampling or whether selected patients can be managed conservatively through active surveillance. Our results demonstrate that PSA density showed clinically useful diagnostic performance among the evaluated variables. Although not a definitive marker on its own, PSA density appears to be a valuable adjunct to imaging-based risk assessment. These findings are supported by recent research proposing nomogram-based models that incorporate PSA density and mpMRI findings to improve diagnostic precision in patients with PSA < 10 ng/mL and PI-RADS $\leq$ 3 lesions.[15] Other large-scale studies have similarly emphasized that PSA density retains value even in lower-suspicion mpMRI categories, particularly when integrated with risk calculators.[16]
In our cohort, PSA density emerged as one of the most informative variables, albeit with moderate performance. Rather than replacing imaging or clinical evaluation, PSA density may serve as a contextual adjunct, particularly in cases in which biopsy indications are ambiguous. This underscores the need for multiparametric approaches that integrate quantitative MRI data, laboratory values, and patient characteristics into risk models. Similar conclusions were drawn by Wen et al., who demonstrated the value of combining PI-RADS v2.1 scores with PSA density to enhance predictive accuracy in patients with PSA levels between 4 and 10 ng/mL.[17] Furthermore, Rajendran et al. highlighted the role of PSA density-based stratification as part of precision tools designed to individualize PCa management.[19] Based on our findings, we believe that PSA density should be interpreted within a multiparametric clinical framework rather than as a standalone indicator for biopsy decision-making.
It should also be considered that most cases of PCa detected in our cohort were low-grade tumors. The inclusion of clinically insignificant PCa in the malignant group may have attenuated the observed associations and reduced the discriminatory performance of PSA density and hematological markers. Because PI-RADS was primarily designed to identify csPCa, the inclusion of low-grade disease may have influenced the diagnostic performance observed in our study.[5] Future studies focusing specifically on csPCa may better define the clinical utility of these markers in patients with PI-RADS 2 lesions.
Among the hematologic indices evaluated in this study, including neutrophil and monocyte counts, NPR, and MPR, several showed statistically significant differences between the benign and malignant groups. However, their low AUC values indicate limited discriminatory ability despite these group-level differences, suggesting that they are not sufficiently robust for independent clinical use. These findings are consistent with previous studies reporting the limited specificity and inconsistent diagnostic performance of systemic inflammatory markers in PCa.[18,20] Although inexpensive and readily available, these markers should be regarded as exploratory adjuncts rather than primary diagnostic tools. Future studies should focus on integrating PSA density with validated biomarkers and multiparametric risk models to improve diagnostic performance. Furthermore, because PPV and NPV are influenced by disease prevalence, the diagnostic performance observed in our selected biopsied cohort should be interpreted with caution and may not be directly generalizable to all patients with PI-RADS 2 lesions.
Although PSA density alone may not provide definitive diagnostic certainty, it may serve as a complementary parameter for risk stratification in patients with borderline mpMRI findings and PSA levels in the gray zone; however, its clinical utility should be confirmed in prospective studies before routine use. Rather than being viewed in isolation, PSA density should be interpreted alongside imaging findings, digital rectal examination results, and individual patient characteristics such as age, comorbidities, and family history. This integrative approach aligns with the movement toward precision urology, aiming to tailor interventions to individual risk profiles while minimizing harm.[19] Looking ahead, PSA density may be further refined through its inclusion in multiparametric nomograms or machine learning-based triage systems that integrate quantitative imaging, laboratory data, and clinical features to enhance biopsy decision-making. Emerging artificial intelligence platforms trained on PI-RADS 2 to 3 lesions are already showing potential to reduce interobserver variability and optimize biopsy decision pathways.[21,22]
Despite guideline recommendations for conservative management, accumulating evidence suggests that up to 5% to 10% of PI-RADS 2 lesions may harbor malignancy.[14,23] This diagnostic ambiguity creates a persistent clinical dilemma, as unnecessary biopsies must be weighed against the risk of underdiagnosis. In this context, refining patient selection remains critical. Although PSA density has emerged as a helpful adjunct, its moderate performance underscores the importance of integrating complementary clinical and imaging parameters into multiparametric risk models. Recent evidence suggests that AI-assisted diagnostic platforms and structured multiparametric frameworks may improve mpMRI interpretation, reduce interobserver variability, and enhance the detection of csPCa, particularly in equivocal PI-RADS 2 to 3 lesions.[22,23] Future research should focus on validating these approaches in prospective cohorts and tailoring algorithms specifically to low-suspicion mpMRI categories such as PI-RADS 2 findings.
This study has several limitations. First, its retrospective, single-center design and inclusion of only patients who underwent prostate biopsy may have introduced selection bias and limited the generalizability of the

findings to all patients with PI-RADS 2 lesions. Because biopsy decisions were based on routine clinical practice rather than predefined study criteria, selection bias cannot be excluded. Second, the relatively small number of prostate cancer cases limited the statistical power and precluded multivariable logistic regression analyses. Third, MRI-targeted biopsy was not performed because all patients had PI-RADS 2 lesions; therefore, the detected cancers may not have originated from the PI-RADS 2 lesions themselves but may have been identified through systematic biopsy. Finally, the exploratory evaluation of multiple hematologic indices without adjustment for multiple comparisons warrants cautious interpretation of the statistically significant findings. Despite these limitations, the study has notable strengths, particularly its exclusive focus on PI-RADS 2 lesions, an underexplored clinical subgroup. By evaluating PSA density and selected hematologic indices in this low-suspicion cohort, our findings provide preliminary evidence that may inform future prospective validation studies.

CONCLUSIONS

PSA density demonstrated moderate discriminatory performance for detecting PCa in this selected cohort of biopsied patients with PI-RADS 2 lesions, with high sensitivity but limited specificity. Although several hematologic indices also showed moderate discriminatory performance, their overall clinical utility appears limited. Larger prospective multicenter studies are warranted to validate these findings and determine their potential role in clinical decision-making.

SUMMARY

PI-RADS 2 MRI findings are usually considered low risk. In this biopsied cohort, PSA density modestly identified PCa, whereas hematologic markers had limited clinical value.

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CONFLICT OF INTEREST

The authors report no conflict of interest.

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DATA AVAILABILITY

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

AUTHOR CONTRIBUTIONS

A.T. and M.K. contributed to the design and implementation of the research. A.T. and Y.A. contributed to the analysis of the results and writing of the manuscript. A.T. and E.A. contributed to editing and reviewing the manuscript. All authors conceived and supervised the project, read the manuscript, and approved the final version.

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