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Combining Systemic Inflammatory Indices with Prostate Volume for Differentiating Prostate Cancer from Benign Prostatic Hyperplasia

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Abstract

Objective

Prostate-specific antigen (PSA) testing is widely used for prostate cancer (PCa) screening; however, its limited specificity remains a major challenge, as elevated PSA levels also occur in benign prostatic hyperplasia (BPH). Systemic inflammatory markers, including neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), and systemic immune-inflammation index (SII), have been proposed as adjunctive diagnostic tools.

Method

This retrospective study included 100 patients (50 PCa and 50 BPH) who underwent transrectal ultrasound-guided prostate biopsy between October 2018 and October 2021. Clinical data, including PSA, prostate volume (PV), and complete blood counts, were collected. Inflammatory indices were calculated and evaluated using receiver operating characteristic (ROC) analysis.

Results

NLR, MLR, RLR, and SII were significantly elevated in PCa patients ($p < 0.05$). However, their individual diagnostic performance was modest (AUC range: 0.624–0.656). The combined inflammatory model showed limited improvement (AUC = 0.662). In contrast, PV demonstrated superior diagnostic accuracy (AUC = 0.774) with high specificity. When combined with inflammatory markers, PV further improved predictive performance, achieving a positive predictive value of 70.3% and a negative predictive value of 73.9%.

Conclusion

Systemic inflammatory markers alone have limited diagnostic value in distinguishing PCa from BPH. However, their combination with prostate volume may enhance diagnostic accuracy and provide a simple, non-invasive approach for risk stratification in clinical practice.

Introduction

Benign prostatic hyperplasia (BPH) and prostate cancer (PCa) are common conditions affecting aging men. While BPH originates predominantly in the transition zone of the prostate, PCa arises mainly from the peripheral zone, with some overlap [1, 2]. Clinical symptoms of both conditions are often similar, making differentiation challenging.

According to the most recent GLOBOCAN 2020 estimates from the International Agency for Research on Cancer, PCa accounted for approximately 1.4 million new cases and remains one of the most commonly diagnosed cancers in men worldwide [3]. Prostate-specific antigen (PSA) remains the standard screening tool for PCa; however, it lacks specificity. Elevated PSA levels are also observed in BPH,

prostatitis, and other non-malignant conditions [4, 5]. Although diagnostic uncertainty is particularly noted in the "gray zone" of PSA (2–10 ng/mL) [6, 7], overlap in PSA values occurs even outside this range. Therefore, supplementary diagnostic markers are needed to improve accuracy across the full spectrum of PSA levels.

Inflammation is increasingly recognized as a contributor to cancer initiation and progression [8, 9]. Hematological indices derived from complete blood counts—such as neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII)—have shown diagnostic and prognostic value in various cancers, including PCa [10–13]. However, their role in distinguishing PCa from BPH remains underexplored.

This study evaluates the utility of six inflammation-based indices and their combinations with prostate volume (PV) in differentiating PCa from BPH in patients with varying PSA levels.

Materials and Methods

Patient Selection

This study was derived from a retrospectively maintained clinical cohort of 116 patients who underwent diagnostic evaluation at the People's Hospital of Xinjiang Uygur Autonomous Region between October 2016 and October 2021. A subset of this cohort (80 patients) was previously analyzed in Guo et al. (Frontiers in Oncology, 2025). The present study addresses different clinical questions and endpoints.

After applying the inclusion criteria, 100 male patients were included in the present analysis, consisting of 50 patients with histologically confirmed prostate cancer (PCa) and 50 patients with benign prostatic hyperplasia (BPH). All diagnoses were confirmed by transrectal ultrasound (TRUS)-guided prostate biopsy. None of the BPH patients showed evidence of malignancy.Serum PSA levels ranged from 0.36 to 1000 ng/mL, representing a real-world spectrum rather than a PSA-restricted “gray-zone” cohort. Although the gray zone (2.5–10 ng/mL) is often emphasized in screening guidelines, this study aimed to examine marker performance across the full diagnostic range. Digital rectal examination (DRE) findings were not consistently available and are acknowledged as a study limitation. Patients who had taken 5-alpha-reductase inhibitors, systemic corticosteroids, or non-steroidal anti-inflammatory drugs within 6 months prior to testing were excluded to avoid confounding effects on PSA and inflammatory markers.

Sample size

Because this was a retrospective exploratory study, all consecutive patients meeting inclusion and exclusion criteria during the study period (n = 100; 50 PCa and 50 BPH) were included. No prospective sample-size calculation was performed. To inform interpretation, a post-hoc power statement for the primary diagnostic metric (prostate volume AUC = 0.774) is provided in the Supplement File 2.

Blood Sampling and Procedures

In our institution, men scheduled for prostate biopsy are admitted for pre-biopsy laboratory testing and imaging. Peripheral blood was drawn on the morning of the second hospital day to minimize diurnal variation. In our institution, venous blood samples for PSA testing are routinely obtained before digital rectal examination to avoid procedure-related transient PSA elevation.

Prostate Volume and Biopsy

Prostate volume (PV) was measured by transrectal ultrasonography using the ellipsoid formula (π/6 × width × length × height). Biopsies were TRUS-guided, and 10–13 cores were obtained per patient.

Inflammatory Indices and Statistics

Inflammation-based indices were calculated as follows: NLR = Neutrophil count / Lymphocyte count. MLR = Monocyte count / Lymphocyte count. PLR = Platelet count / Lymphocyte count. RPR = RDW / Platelet count. RLR = RDW / Lymphocyte count. SII = (Neutrophil count × Platelet count) / Lymphocyte count. PV was calculated using the ellipsoid formula: π/6 × width × length × height.

This study was reported in accordance with the TRIPOD (Transparent Reporting of a multivariable prediction model for Individual Prognosis Or Diagnosis) guideline. A completed TRIPOD checklist is provided in Supplementary File S1.

Statistical Analysis

Statistical analysis was performed using GraphPad Prism (GraphPad Software, San Diego, CA). ROC curve analysis was used to determine diagnostic accuracy. Sensitivity, specificity, AUC, PPV, and NPV were calculated. Logistic regression was applied to assess the combined predictive value of multiple markers. A two-tailed p-value < 0.05 was considered statistically significant.

For completeness, post-hoc power for the primary ROC analysis (testing AUC > 0.5 at α = 0.05 with n = 50 cases and n = 50 controls) was computed using the Hanley–McNeil standard error; for PV AUC = 0.774 the estimated power was ≈ 0.9999 (SE = 0.0469; 95% CI 0.682–0.866) (Supplementary File S2).

Results

Baseline Characteristics

Table 1 and Supplementary Figure S3 summarize the baseline characteristics of patients. The mean age was comparable between groups. PSA levels in PCa patients ranged from 4.98 to > 1000 ng/mL (median: 107.37 ng/mL), significantly higher than in BPH patients (median: 7.94 ng/mL). PV was significantly smaller in the PCa group (p < 0.001) (Table 2).

Table 1
Baseline characteristics of study participants.

Characteristic	BPH (n = 50)	PCa (n = 50)	P Value
Mean age (range), yr	63.34 (51 ~ 76)	61.6 (49 ~ 82)	0.2111
Median tPSA (range), ng/ml	7.94 (0.14 ~ 71.39)	107.37 (4.98 ~ 1000)	< 0.0001
Median fPSA (range), ng/ml	1.78 (0.03 ~ 22.82)	8.705 (0.93 ~ 30)	< 0.0001
Median f/t PSA (range), ng/ml	0.252 (0.1 ~ 0.65)	0.161 (0.03 ~ 0.45)	< 0.0001
Abbreviations: tPSA = total prostate-specific antigen; fPSA = free prostate-specific antigen;			

Table 2
Comparison of laboratory parameters and prostate volume measurements between patients with benign prostatic hyperplasia (BPH) and prostate cancer (PCa).

Characteristic	BPH (n = 50) Mean ± SE	PCa (n = 50) Mean ± SE	P-value
WBC, $\times 10^9$/L	7.0 ± 0.275	7.0 ± 0.313	0.4860
Hb, g/L	144.72 ± 2.882	138.6 ± 3.573	0.1856
Neutrophils, $\times 10^9$/L	4.24 ± 0.209	4.81 ± 0.277	0.1015
Lymphocytes, $\times 10^9$/L	1.85 ± 0.093	1.60 ± 0.091	0.0532
Red cell distribution width (RDW), %	13.18 ± 0.122	13.13 ± 0.132	0.8155
Monocytes, $\times 10^9$/L	0.48 ± 0.026	0.50 ± 0.028	0.4961
Platelet, $\times 10^9$/L	224.22 ± 8.285	221.34 ± 8.891	0.8132
Median PV, ml	68.81 ± 5.692	37.92 ± 2.965	< 0.0001
Abbreviations: Prostate Volume = PV.			

Inflammatory Markers

NLR, MLR, RLR, and SII levels were significantly elevated in PCa patients compared to BPH ($p < 0.05$) (Fig. 1A). No significant differences were observed in PLR or RPR(Fig. 1B). Inflammatory markers showed no significant association with tumor grade (Gleason score) (Table 3).

Table 3. Inflammatory Marker Levels (NLR, MLR, SII) and Their Distribution Among Gleason Score Categories in PCa.

		Percentage of men		
Variable	patients(n)	Gleason Score 6	Gleason Score 7	Gleason Score ≥ 8
NLR	50			
Low	21	14.28%	42.86%	42.86%
High	29	20.7%	31.03%	48.27%
MLR	50			
Low	19	21.05%	26.32%	52.63%
High	31	16.12%	41.94%	41.94%
SII	50			
Low	24	16.68%	41.66%	41.66%
High	26	19.23%	30.77%	50%

Abbreviations: NLR = neutrophil-to-lymphocyte ratio; MLR = monocyte-to-lymphocyte ratio; PLR = platelet-to-lymphocyte ratio; SII = systemic immune-inflammation index;

ROC Curve Analysis

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic accuracy of individual inflammatory markers and their combinations in distinguishing PCa from BPH (Table 4). Among the six indices analyzed, NLR, MLR, and SII showed statistically significant but moderate diagnostic performance:

Table 4

A: Comparison of diagnostic accuracy between single predictors and combined predictive models.

Variables	AUC	Cut off	Sensitivity	Specificity	95% interval confidence	P-value
NLR	0.656	2.77	58%	76%	0.548–0.764	0.0072
MLR	0.624	0.27	62%	58%	0.515–0.733	0.0323
SII	0.639	673.75	52%	78%	0.529–0.865	0.0167
PV	0.774	32.23	50%	94%	0.683–0.865	<0.0001
NLR + MLR	0.662	-	-	-	0.556–0.768	0.0052
NLR + SII	0.656	-	-	-	0.548–0.764	0.0072
MLR + SII	0.644	-	-	-	0.536–0.753	0.0131
NLR + MLR+SII	0.662	-	-	-	0.555–0.768	0.0054
PV + NLR+MLR + SII	0.788	-	-	-	0.699–0.877	<0.0001

Receiver operating characteristic (ROC) curve analysis was used to estimate the area under the curve (AUC) with 95% confidence intervals Abbreviations: AUC = area under the curve; CI = confidence interval; PSA = prostate-specific antigen; PV = prostate volume; NLR = neutrophil-to-lymphocyte ratio; MLR = monocyte-to- lymphocyte ratio

Table 4

B. Positive and negative predictive values of combined models for differentiating prostate cancer from benign prostatic hyperplasia

Variables	PPV	NPV
NLR + MLR	65%	60%
NLR + SII	66.67%	60.66%
MLR + SII	65.12%	61.40%
NLR + MLR+SII	66.67%	62.07%
PV + NLR+MLR + SII	70.37%	73.91%

PPV= positive predictive value; NPV=negative predictive value. Predictive values were derived from logistic regression models.

NLR had an AUC of 0.656 (95% CI: not specified, p = 0.0072), indicating fair discrimination. MLR demonstrated an AUC of 0.6242 (p = 0.0323). SII showed an AUC of 0.6388 (p = 0.0167) (Fig. 2). The combined use of NLR, MLR, and SII slightly improved diagnostic performance, with an AUC of 0.6616 (95% CI: 0.555–0.7682), a positive predictive value (PPV) of 66.67%, and a negative predictive value (NPV) of 62.07% (Table 4 and Fig. 3). However, this improvement was not statistically significant compared to the individual markers.

In contrast, prostate volume (PV) alone yielded a higher AUC of 0.7740, with strong specificity but lower sensitivity. When PV was combined with the three inflammatory markers (NLR, MLR, and SII), the overall diagnostic performance improved, resulting in a PPV of 70.3% and an NPV of 73.91%, suggesting that the combination model incorporating PV and inflammatory markers achieved the highest discrimination (AUC = 0.7876). (Table 4A,B).

Discussion

Prostate cancer (PCa) remains a major global health challenge and is the second most frequently diagnosed cancer among men worldwide [14]. Although the prostate-specific antigen (PSA) test has long served as a standard screening tool, its diagnostic limitations are well recognized, particularly in differentiating PCa from benign prostatic hyperplasia (BPH). Elevated PSA levels may occur in both benign and malignant conditions, leading to considerable diagnostic overlap. This challenge is especially pronounced within the “gray zone” (PSA 2.5–10 ng/mL), where PCa and BPH frequently present with similar PSA values [6, 7]. In the present study, we evaluated systemic inflammatory markers together with prostate volume (PV) for distinguishing PCa from BPH across the full PSA spectrum. Unlike many previous studies that focused primarily on the PSA gray zone, our cohort included patients with PSA values ranging from 0.36 to 1000 ng/mL, reflecting real-world diagnostic heterogeneity encountered in routine clinical practice.

To address the limitations of PSA testing alone, increasing attention has been directed toward identifying adjunctive biomarkers that can improve diagnostic accuracy. Inflammation is now widely recognized as an important contributor to tumor development and progression. Chronic inflammatory processes may contribute to tumor initiation and the creation of a pro-tumorigenic microenvironment [8, 9]. Inflammation-based hematologic indices derived from routine blood tests such as the neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), and systemic immune-inflammation index (SII) have recently emerged as potential diagnostic and prognostic biomarkers in several malignancies, including PCa [10–12, 15–19]. In the present study, NLR, MLR, SII, and RLR were significantly elevated in patients with PCa compared with those with BPH, suggesting the presence of systemic immune dysregulation associated with tumor development. These findings are biologically plausible. Neutrophils can facilitate tumor progression through the release of extracellular traps and pro-angiogenic factors, while suppressing cytotoxic lymphocyte activity [20]. In parallel, circulating monocytes may differentiate into tumor-associated macrophages (TAMs), which are known to promote tumor invasion, angiogenesis, and immune evasion [21, 22].

Despite these biological associations, the diagnostic performance of individual inflammatory markers in our study was modest. ROC analysis showed AUC values ranging from 0.624 to 0.656 for NLR, MLR, and SII, indicating limited discriminatory capacity when used alone. These findings are consistent with previous reports, such as the study by Lanyu Wang et al. [23], which also demonstrated moderate diagnostic accuracy for similar inflammatory indices. However, an important observation of the present study is that combining inflammatory markers with prostate volume improved overall diagnostic

performance. While inflammatory indices alone provided only moderate discrimination, the integration of PV increased predictive performance, highlighting the potential value of combining systemic inflammatory indicators with anatomical parameters.

Prostate volume emerged as the strongest individual discriminator in our cohort, with an AUC of 0.774. Patients with PCa had significantly smaller prostate volumes than those with BPH, which is consistent with the known anatomical distribution of these conditions: BPH predominantly involves the transition zone, whereas PCa most commonly originates in the peripheral zone. When PV was combined with inflammatory markers, predictive performance further improved (PPV 70.3%, NPV 73.91%), suggesting that integrating structural and systemic inflammatory information may provide a more comprehensive approach to pre-biopsy risk assessment. Importantly, both prostate volume measurement and blood-derived inflammatory markers are readily available in routine clinical practice. Their combined use may therefore represent a simple, non-invasive, and cost-effective strategy for improving clinical risk stratification in patients undergoing evaluation for suspected PCa.

Although absolute leukocyte counts were generally within normal reference ranges, ratio-based indices can reveal subtle immune disequilibrium that may not be detected by conventional hematologic parameters. Additional inflammatory biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α) could provide further insight into systemic inflammatory status but were not available in the present retrospective dataset. Similarly, BMI and detailed comorbidity data were incomplete and could not be analyzed.

While these inflammatory markers alone are unlikely to replace established diagnostic tools such as PSA testing, imaging, or biopsy, they may serve as useful adjunctive indicators in clinical decision-making. Because these indices are inexpensive and derived from routine blood tests, they may be particularly valuable in resource-limited settings or as part of preliminary risk assessment prior to biopsy.

Several limitations should be acknowledged. First, this study was retrospective and conducted at a single center, which may introduce selection bias. Second, the sample size was relatively small, which may limit statistical power and generalizability. Third, potential confounding factors such as subclinical infections, inflammatory diseases, or medication use were not fully controlled. Future prospective multicenter studies with larger cohorts are needed to validate these findings and to explore the integration of inflammatory markers with imaging and molecular biomarkers.

In summary, this study demonstrates that systemic inflammatory markers—particularly NLR, MLR, and SII—are significantly elevated in patients with PCa compared with those with BPH. Although their individual diagnostic accuracy is modest, their combination with prostate volume improves overall predictive performance. These findings support the potential value of integrating readily available inflammatory indices with anatomical parameters to enhance non-invasive risk stratification before prostate biopsy.

Declarations

Disclosure Statement

The authors have nothing to disclose.

Funding

This research received no external funding.

Ethics Statement

This retrospective study was approved by the Medical Ethics Committee of People's Hospital of Xinjiang Uygur Autonomous Region, Xinjiang, China. The study was conducted in accordance with the Declaration of Helsinki. The requirement for informed consent was waived due to the retrospective design and the use of fully anonymized clinical data. No direct patient identifiers were collected, and no patient contact or recruitment was involved, resulting in minimal risk to participants.

Informed Consent Statement

The need forinformed consent was formally waived by the Medical Ethics Committee of the People's Hospital of Xinjiang Uygur Autonomous Region.

Registry and the Registration No. of the study: N/A

Generative AI statement

Generative artificial intelligence tools (ChatGPT, OpenAI, GPT-4) were used for language editing and improvement of grammar and clarity. The authors take full responsibility for the scientific content, data interpretation, and conclusions of the manuscript.

Data Availability Statement

The datasets generated and/or analyzed during the current study are not publicly available due to patient privacy and institutional restrictions but are available from the corresponding author on reasonable request.

Animal Studies: N/A

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Figures

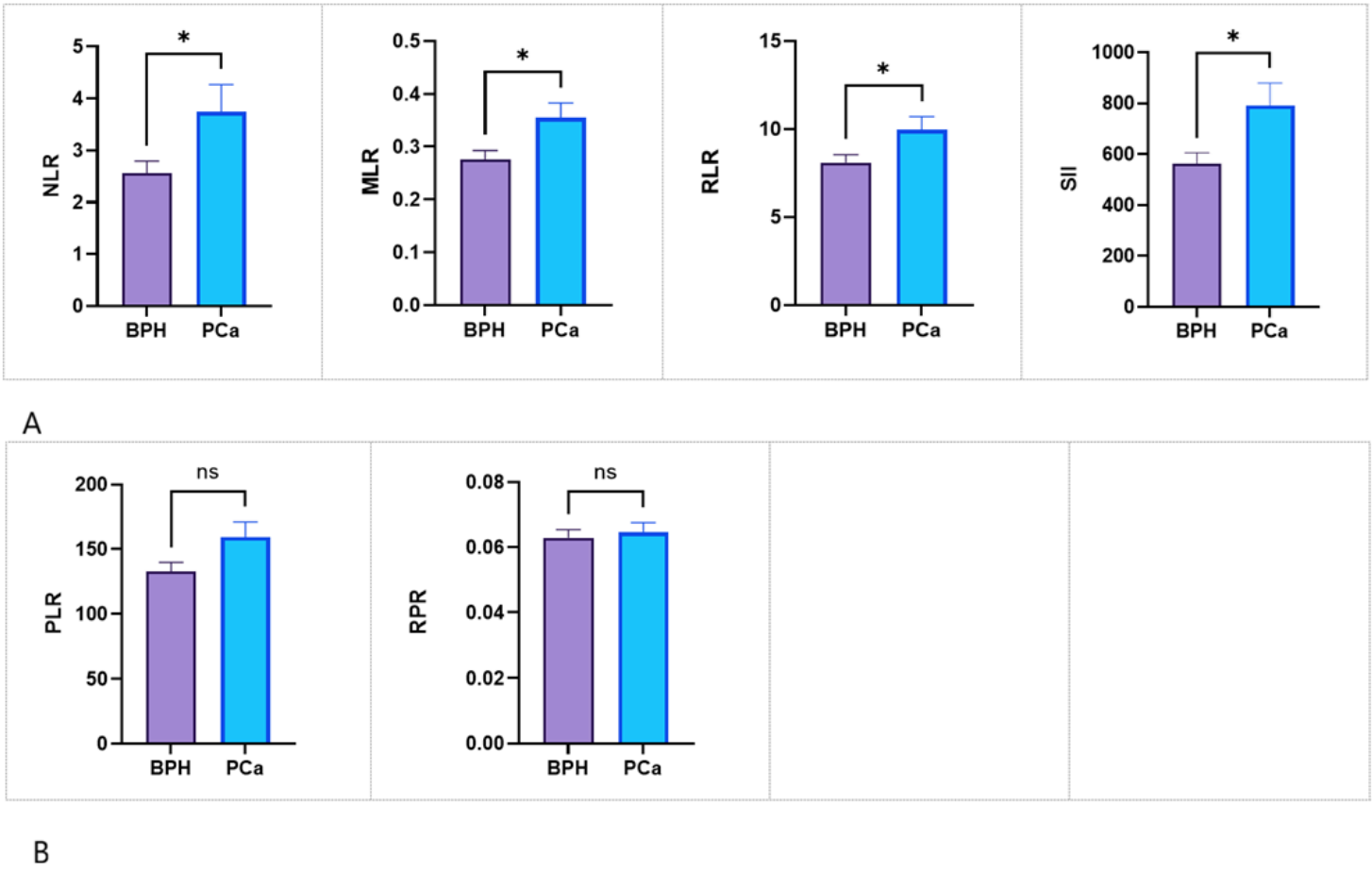


Figure 1

A. Comparison of inflammation-derived indicates between BPH and PCa patients. Neutrophil-to-lymphocyte ratio (NLR); Monocyte-to- lymphocyte ratio (MLR); RDW -to-lymphocyte ratio (RLP) and

systemic immune-inflammation index (SII; neutrophils x platelets)/lymphocytes). P-values: Student's t-test (parametric). *p<0.05, **p<0.01

B. Comparison of platelet-to- lymphocyte ratio (PLR) and red cell distribution width (RDW)-to- platelet ratio (RPR) between BPH and PCa patients. ns = not significant (P ≥ 0.05)

Figure2

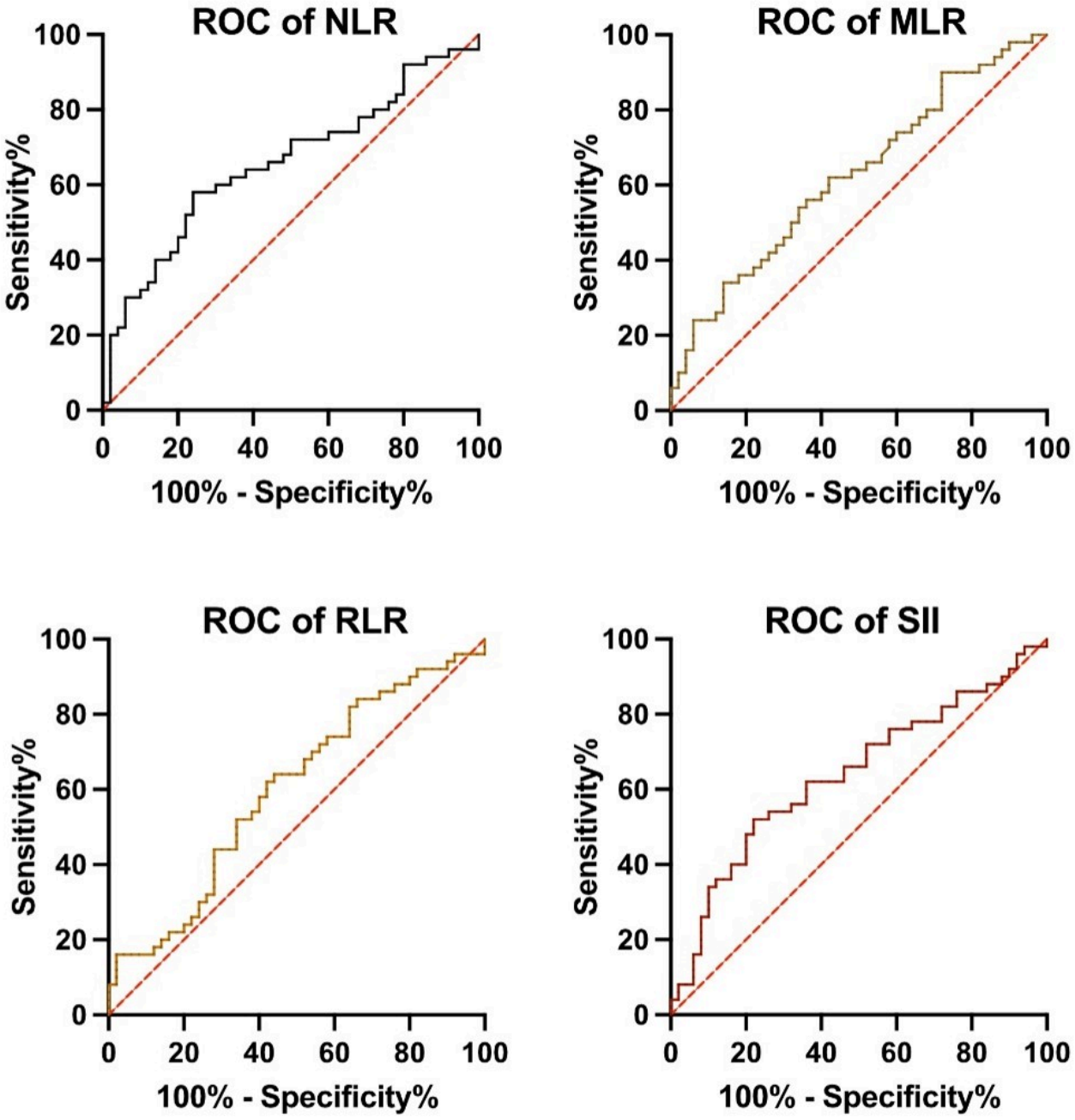


Figure 2

The receiver operating characteristic (ROC) curves of NLR—neutrophil/lymphocyte ratio, LMR—lymphocyte/monocyte ratio, (C) RLR—Red cell distribution width/lymphocytes ratio, and (D) SII—systemic immune inflammation index. AUC, area under the ROC curve.

Figure3

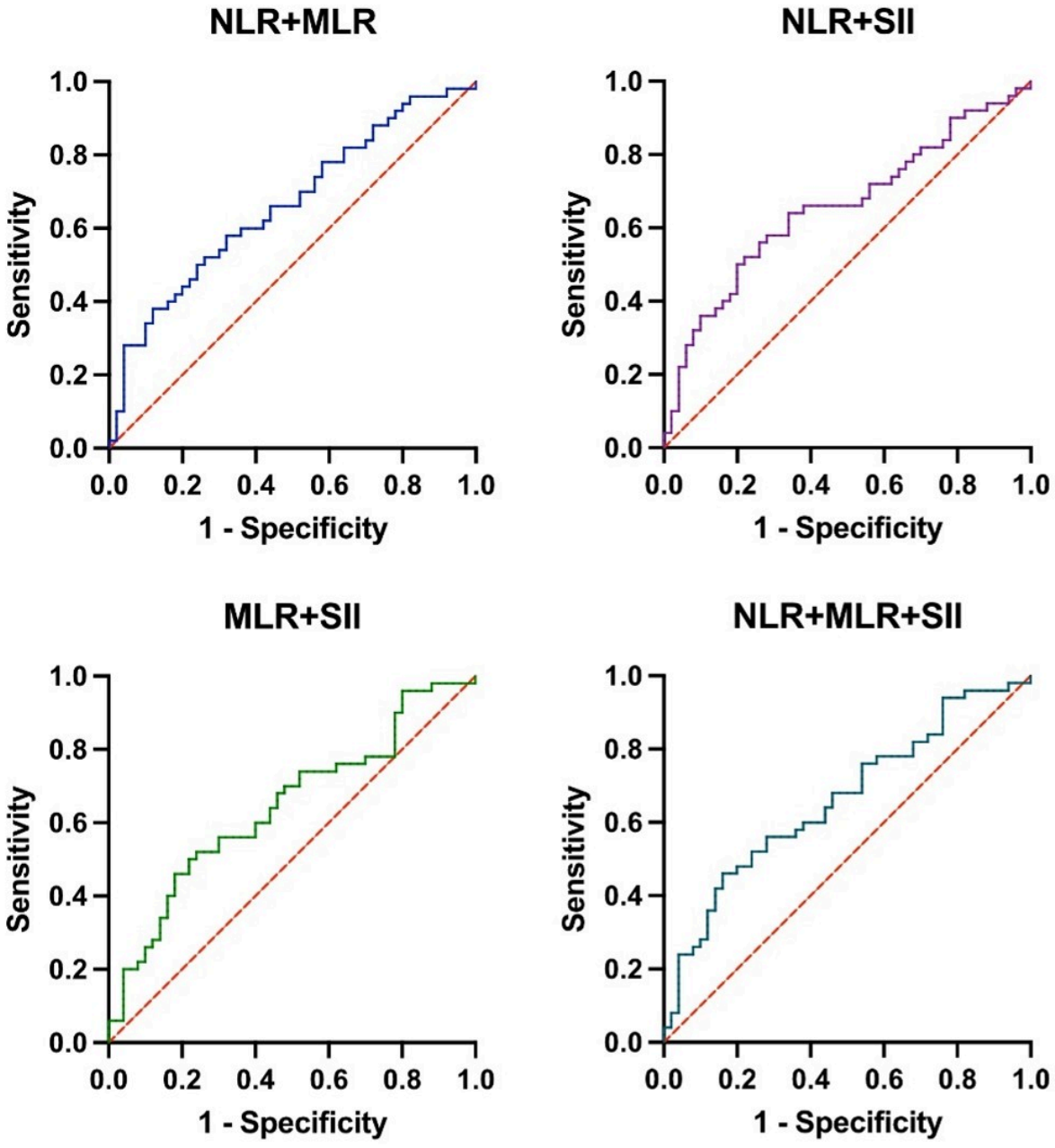


Figure 3

The diagnostic efficiency of the NLR, MLR, and SII in combination for PCa was analyzed by ROC curves. ROC, receiver operating characteristic; AUC, area under the ROC curve.

Supplementary Files

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

- SupplementaryFileS2PosthocPowerAnalysis.docx