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Added Value of Multiparametric Magnetic Resonance Imaging to Clinical Nomograms for Predicting Adverse Pathology in Prostate Cancer

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Abstract

Purpose: We examined the additional value of preoperative prostate multiparametric magnetic resonance imaging and transrectal ultrasound/multiparametric magnetic resonance imaging fusion guided targeted biopsy when performed in combination with clinical nomograms to predict adverse pathology at radical prostatectomy.

Materials and Methods: We identified all patients who underwent 3 Tesla multiparametric magnetic resonance imaging prior to fusion biopsy and radical prostatectomy. The Partin and the MSKCC (Memorial Sloan Kettering Cancer Center) preradical prostatectomy nomograms were applied to estimate the probability of organ confined disease, extraprostatic extension, seminal vesicle invasion and lymph node involvement using transrectal ultrasound guided systematic biopsy and transrectal ultrasound/multiparametric magnetic resonance imaging fusion guided targeted biopsy Gleason scores. With radical prostatectomy pathology as the gold standard we developed multivariable logistic regression models based on these nomograms before and after adding multiparametric magnetic resonance imaging to assess any additional predictive ability.

Results: A total of 532 patients were included in study. When multiparametric magnetic resonance imaging findings were added to the systematic biopsy based MSKCC nomogram, the AUC increased by 0.10 for organ confined disease ($p < 0.001$), 0.10 for extraprostatic extension (p

= 0.003), 0.09 for seminal vesicle invasion (p = 0.011) and 0.06 for lymph node involvement (p = 0.120). Using Gleason scores derived from targeted biopsy compared to systematic biopsy provided an additional predictive value of organ confined disease (Δ AUC 0.07, p = 0.003) and extraprostatic extension (Δ AUC 0.07, p = 0.048) at radical prostatectomy with the MSKCC nomogram. Similar results were obtained using the Partin nomogram.

Conclusions: Magnetic resonance imaging alone or in addition to standard clinical nomograms provides significant additional predictive ability of adverse pathology at the time of radical prostatectomy. This information can be greatly beneficial to urologists for preoperative planning and for counseling patients regarding the risks of future therapy.

Keywords

prostatic neoplasms; prostatectomy; image-guided biopsy; risk assessment; nomograms

Accurate PCa preoperative staging is essential for patient counseling as well as treatment planning. For example, in cases of suspected EPE or SVI external beam radiation therapy may be preferred over RP or brachytherapy due to the risks of incomplete resection or under dosing, respectively.[1] Preoperative knowledge of EPE may also inform surgical management, potentially allowing for modified surgical techniques such as a wider resection margin or even nerve resection to maximize oncologic efficacy and minimize the risk of positive surgical margins.[2]

Prediction models that combine clinical stage, serum PSA levels and Gleason grade in the biopsy specimens are commonly used in clinical practice to predict the pathological stage of PCa and, thus, aid in preoperative decision making. The Partin nomogram[3] and the MSKCC preradical prostatectomy nomogram[4] are examples of validated predictive tools that are widely used for patient counseling.

Multiparametric MRI offers multiple advantages that can be incorporated into existing nomograms, including imaging data on multiple adverse features and the ability to perform Tbx to detect more clinically significant cancer. There is a clear benefit of mpMRI and Tbx for diagnosing clinically significant disease over that of Sbx alone. Siddiqui et al found that Tbx can detect up to 30% more high grade disease and less indolent cancer compared with Sbx.[5] Ahmed et al found that mpMRI had greater sensitivity than standard TRUS (up to 92% vs up to 60%) in the ability to rule out clinically significant disease.[6] However, it is uncertain whether the upgrading that occurs due to mpMRI guided biopsies helps predict adverse pathology at surgery.

Due to the high costs of MRI and the high prevalence of PCa mpMRI poses a significant financial burden on society.[7–10] As a result there is still debate regarding the role of MRI for the preoperative evaluation of PCa. Guidelines from the AUA (American Urological Association),[11] the EAU (European Association of Urology)[12] and the NCCN® (National Comprehensive Cancer Network®)[13] suggest a role for MRI, especially in the setting of high risk disease, but they do not provide definite indications for application.

Many clinically used nomograms have been validated using standard TRUS guided biopsies. However, studies of the additional value of prostate mpMRI and Tbx to validated clinical nomograms are scarce.[14–17] In this study we examined the additional value of prostate mpMRI data and Tbx pathology data to the MSKCC[4] and Partin[3] nomograms to predict adverse pathological features on final pathology. In addition, due to the increased performance of Tbx in clinical practice we sought to determine whether using pathology data from Tbx compared to Sbx would provide additional benefit to predict these adverse pathological features on RP in these nomograms.

MATERIALS AND METHODS

Patient Selection

All patients were enrolled in an institutional review board approved protocol and provided informed consent. A prospectively maintained database was queried for all patients who underwent mpMRI, Sbx and/or Tbx and RP from May 2007 to September 2017. We collected baseline demographics, including age, PSA, digital rectal examination, biopsy and mpMRI findings, including the NIH suspicion score, EPE, SVI and the largest lesion diameter.

PI-RADS™ v2 has been in use at our institution since May 2015 along with the previously validated NIH suspicion score.[18] However, in the current study we did not use PI-RADS v2 data since patients who underwent mpMRI prior to May 2015 did not undergo prospective mpMRI interpretation based on PI-RADS v2. Patients with insufficient mpMRI or biopsy data were excluded from study. Any patients with prior radiation or hormonal therapy were also excluded.

Staging Nomograms

The likelihood of OCD, EPE, SVI and LNI according to the 2010 Partin nomogram[3] and the MSKCC preradical prostatectomy nomogram[4] were recorded based on pretreatment characteristics, including PSA level, Gleason Grade Group and clinical stage. Sbx as well as Tbx results were used with each of these nomograms.

Imaging and Biopsy Protocol

Diagnostic mpMRI of the prostate was performed with a 3 Tesla Achieva scanner (Philips, Cleveland, Ohio) using a Medrad® BPX-30 endorectal coil and a 16-channel SENSE cardiac surface coil (Philips) as previously described.[19] Prostate mpMRIs were evaluated prospectively by 2 radiologists with extensive prostate MRI experience. In most cases mpMRI incorporated triplanar T2-weighted, diffusion weighted, dynamic contrast-enhanced and magnetic resonance spectroscopy sequences. These sequences were combined to produce a 3-point NIH PCa suspicion score of 1—low, 2—moderate or 3—high as previously validated.[18]

Patients then underwent an outpatient prostate biopsy session, which included Tbx of all MRI suspicious lesions with a minimum of 2 cores sampled in the axial and sagittal planes using an end fire TRUS probe.[20] Additionally, all patients underwent Sbx at the same time.

All biopsy and RP pathology findings were reviewed by a single genitourinary pathologist. Robot-assisted RP was performed by a single urologist.

Statistical Analysis

Multivariable logistic regression was done to estimate the association of clinical variables and mpMRI findings with the prediction of adverse pathological features. Using the Sbx and Tbx results we calculated the Partin[3] and MSKCC[4] nomogram estimates of OCD, EPE, SVI and LNI, and incorporated these estimates into multivariable modeling to create a ROC curve. Multiparametric MRI results were then added to each model and the multivariable modeling was reassessed.

The AUC values before and after adding mpMRI results to each model were compared by the Delong method. Nomogram predictions using Sbx and Tbx results were also compared. Statistical significance was considered at $p < 0.05$. All analyses were calculated with Stata/IC™ 13.

RESULTS

Of the 552 patients who underwent RP 20 were excluded from study due to insufficient data or prior PCa treatment. All remaining 532 patients underwent preoperative mpMRI prior to RP and were included in study. Of these patients 327 underwent Tbx plus Sbx and the remaining 205 underwent only Sbx. On the final pathology evaluation EPE, SVI and LNI were found in 115 (21.6%), 37 (7.0%) and 31 patients (6.2%), respectively.

Table 1 summarizes the distribution of preoperative clinical variables. Median age in this group was 61 years (IQR 56–66) and median PSA was 6.2 ng/ml (IQR 4.3–9.9). Of the patients 101 (19.0%) had suspected EPE, 24 (4.5%) had suspected SVI and none had suspected LNI on mpMRI. In the overall cohort the mean ± SD largest prostate lesion diameter was 1.6 ± 0.7 cm on mpMRI.

The mpMRI findings were then incorporated into the MSKCC[4] and Partin[3] nomograms with Sbx results using multivariable logistic regression (supplementary table, http://jurology.com/). These models incorporated mpMRI findings of the NIH suspicion score, EPE, SVI and the largest lesion diameter. A smaller lesion diameter on mpMRI was the strongest predictor of OCD in the Sbx Partin plus mpMRI model (OR 0.64, $p = 0.046$) and in the Sbx MSKCC plus mpMRI model (OR 0.63, $p = 0.039$).

When looking at nonorgan confined disease, mpMRI findings yielded more predictive ability than either of the 2 nomograms alone. The largest lesion diameter on mpMRI was the strongest predictor of EPE at RP in the Sbx Partin[3] plus mpMRI model and the Sbx MSKCC[4] plus mpMRI model (OR 2.33, $p < 0.001$ and OR 2.07, $p = 0.001$, respectively). The largest lesion diameter on mpMRI was also the strongest predictor of LNI at RP in the Sbx Partin plus mpMRI model and the Sbx MSKCC plus mpMRI model (OR 2.10, $p = 0.017$ and OR 2.09, $p = 0.013$, respectively). SVI on mpMRI was the strongest predictor of SVI on RP in the Sbx Partin plus mpMRI model and in the Sbx MSKCC plus mpMRI model (OR 28.10, $p < 0.001$ and OR 35.86, $p < 0.001$, respectively).

Table 2 shows a comparison of MSKCC nomogram[4] AUCs before and after adding mpMRI results. The combined mpMRI and Sbx MSKCC model outperformed the Sbx MSKCC nomogram alone. When comparing the Sbx MSKCC and the Sbx MSKCC plus mpMRI predictive models, the AUC was 0.70 vs 0.80 (p <0.001) for OCD, 0.70 vs 0.80 (p = 0.003) for EPE, 0.81 vs 0.90 (p = 0.011) for SVI and 0.82 vs 0.88 (p = 0.120) for LNI. Similarly the combined mpMRI and Tbx MSKCC model outperformed the Tbx MSKCC nomogram alone. When comparing the Tbx MSKCC and Tbx MSKCC plus mpMRI predictive models, the AUC was 0.77 vs 0.82 (p = 0.046) for OCD, 0.77 vs 0.82 (p = 0.045) for EPE, 0.76 vs 0.85 (p = 0.065) for SVI and 0.88 vs 0.94 (p = 0.023) for LNI.

A similar analysis was done to compare the AUCs of the Partin nomogram[3] before and after adding mpMRI results (table 2). The combined mpMRI and Sbx Partin model outperformed the Sbx Partin nomogram alone. When comparing the Sbx Partin and Sbx Partin plus mpMRI predictive models, the AUC was 0.73 vs 0.81 (p = 0.001) for OCD, 0.66 vs 0.80 (p <0.001) for EPE, 0.80 vs 0.88 (p = 0.022) for SVI and 0.85 vs 0.91 (p = 0.071) for LNI. Similarly the combined mpMRI and Tbx Partin model outperformed the Tbx Partin nomogram alone. When comparing the Tbx Partin and Tbx Partin plus mpMRI predictive models, the AUC was 0.78 vs 0.83 (p = 0.030) for OCD, 0.67 vs 0.79 (p = 0.003) for EPE, 0.83 vs 0.87 (p = 0.452) for SVI and 0.89 vs 0.93 (p = 0.085) for LNI.

The figure shows a comparison of the Sbx and Tbx MSKCC[4] and Partin[3] nomogram ROC curves before and after adding mpMRI for the overall prediction of nonorgan confined disease, including EPE, SVI or LNI.

Finally, the Tbx and Sbx nomograms were compared by ROC analysis. When comparing the Tbx MSKCC and Sbx MSKCC nomograms,[4] the AUC was 0.77 vs 0.70 (p = 0.003) for OCD, 0.77 vs 0.70 (p = 0.048) for EPE, 0.76 vs 0.81 (p = 0.820) for SVI and 0.88 vs 0.82 (p = 0.174) for LNI. When comparing the Tbx Partin and Sbx Partin nomograms,[3] the AUC was 0.78 vs 0.73 (p=0.009) for OCD, 0.67 vs 0.66 (p = 0.230) for EPE, 0.83 vs 0.80 (p = 0.214) for SVI and 0.89 vs 0.85 (p = 0.374) for LNI.

DISCUSSION

In the setting of Sbx our results demonstrated that mpMRI provides additional value to theMSKCC[4] and Partin[3] nomograms to predict adverse pathology at RP. Adding mpMRI findings to these staging nomograms significantly improved the prediction of OCD, EPE and SVI on final pathology. A nonsignificant increase in LNI prediction was seen, which may have been due to the relatively low event rate of LNI as well as the low sensitivity of mpMRI for LNI detection.[21–23]

While it is important that new tests and imaging modalities be assessed in relation to existing tests or nomograms, studies exploring the additional value of mpMRI to these nomograms are scarce and not all in agreement.[15,16] In contrast to our series, in a retrospective study Morlacco et al compared the predictive accuracy of MRI and clinical models, including the Partin nomogram[3] and the CAPRA (Cancer of the Prostate Risk Assessment) score, for PCa staging and found significantly improved accuracy after adding

MRI to the models.[15] One of the main differences between the 2 studies is that in the study by Morlacco et al the overall cohort had more advanced disease with a 42.3%, 30.7% and 16.0% rate of EPE, SVI and LNI compared to 21.6%, 7.0% and 6.2%, respectively, in our study. We found rates of EPE, SVI and LNI similar to those in previously published studies.[2,17,24,25] In the study by Morlacco et al the greater ratio of patients with adverse pathology findings may have been due to mpMRI ordered based on higher clinical suspicion of adverse pathology rather than to a standardized protocol in every patient.[15]

The additional value obtained by adding mpMRI results to the Tbx based nomograms was statistically significant for predicting OCD, EPE and LNI using the MSKCC nomogram[4] and for OCD and EPE prediction using the Partin nomogram.[3] However, the additional value obtained by adding mpMRI results to the Tbx based nomograms was generally not as pronounced as when using the combined mpMRI Sbx nomogram models. The higher Gleason score in targeted biopsies may have been due to incorporating the higher risk into the nomograms, resulting in less significant improvements in the value of adding further mpMRI data to preoperative predictions.

In a recent study of 236 patients Weaver et al found that prostate MRI added no additional value to the MSKCC nomogram[4] for PCa staging.[16] It is not clear from that study whether Tbx or Sbx results were used for risk prediction with the MSKCC nomogram, which we observed was an important distinction. Other differences in the 2 studies include the involvement of 9 radiologists for reading mpMRI in the study by Weaver et al[16] compared to only 2 radiologists in our series. The increased number of radiologists likely negatively impacted overall MRI accuracy as prior research has shown substantial interobserver variability in prostate MRI interpretation.[26,27] Additionally, the Weaver et al cohort had a greater rate of advanced disease than expected based on previous studies with a 35%, 14% and 8% rate of EPE, SVI and LNI, respectively.[16]

Finally, using pathology derived from Tbx compared to Sbx provided significant additional value for predicting OCD on RP when applying the Partin[3] and MSKCC[4] nomograms, and for predicting EPE when applying MSKCC nomogram only. The increased predictive ability of EPE when using Tbx compared to Sbx in the MSKCC but not the Partin[3] nomogram may have been due to the fact that the MSKCC nomogram incorporates additional variables, including the percent of positive cores on biopsy as a surrogate for tumor volume, which would be improved on Tbx, while the Partin nomogram does not incorporate additional variables.[5,28] This suggests a role for updated versions of commonly used clinical nomograms in the era of mpMRI and Tbx.

Our study has several limitations, including the fact that it was a single center study. Additionally, PI-RADS v2 was not used to assess mpMRI uniformly since this study spanned the introduction of PI-RADS v2. Instead, a validated 3-point system was used which was shown to behave similarly to PI-RADS v2.[29] Our study also had a small sample size compared to the original Partin[3] and MSKCC[4] nomograms, which included more than 1,000 patients each. However, with 532 patients we report one of the largest studies of the additional value of mpMRI to clinical staging nomograms.

CONCLUSIONS

Multiparametric MRI alone or in addition to existing validated risk stratification tools, including the Partin[3] and the MSKCC[4] nomograms, provides significant additional predictive ability for adverse pathological features at the time of RP. Updated versions of commonly used clinical nomograms reflecting mpMRI and Tbx results may be necessary to better reflect preoperative risk stratification. By using this information urologists can better counsel patients regarding this outcome after surgery and the potential need for any future therapies.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations and Acronyms

EPE	extraprostatic extension
LNI	lymph node involvement
mpMRI	multiparametric MRI
MRI	magnetic resonance imaging
MSKCC	Memorial Sloan Kettering Cancer Center
NIH	National Institutes of Health
OCD	organ confined disease
PCa	prostate cancer
PI-RADS™	Prostate Imaging Reporting and Data System
PSA	prostate specific antigen
RP	radical prostatectomy
Sbx	systematic TRUS guided biopsy
SVI	seminal vesicle invasion
Tbx	TRUS/mpMRI fusion guided targeted biopsy

TRUS	transrectal ultrasound
v2	version 2

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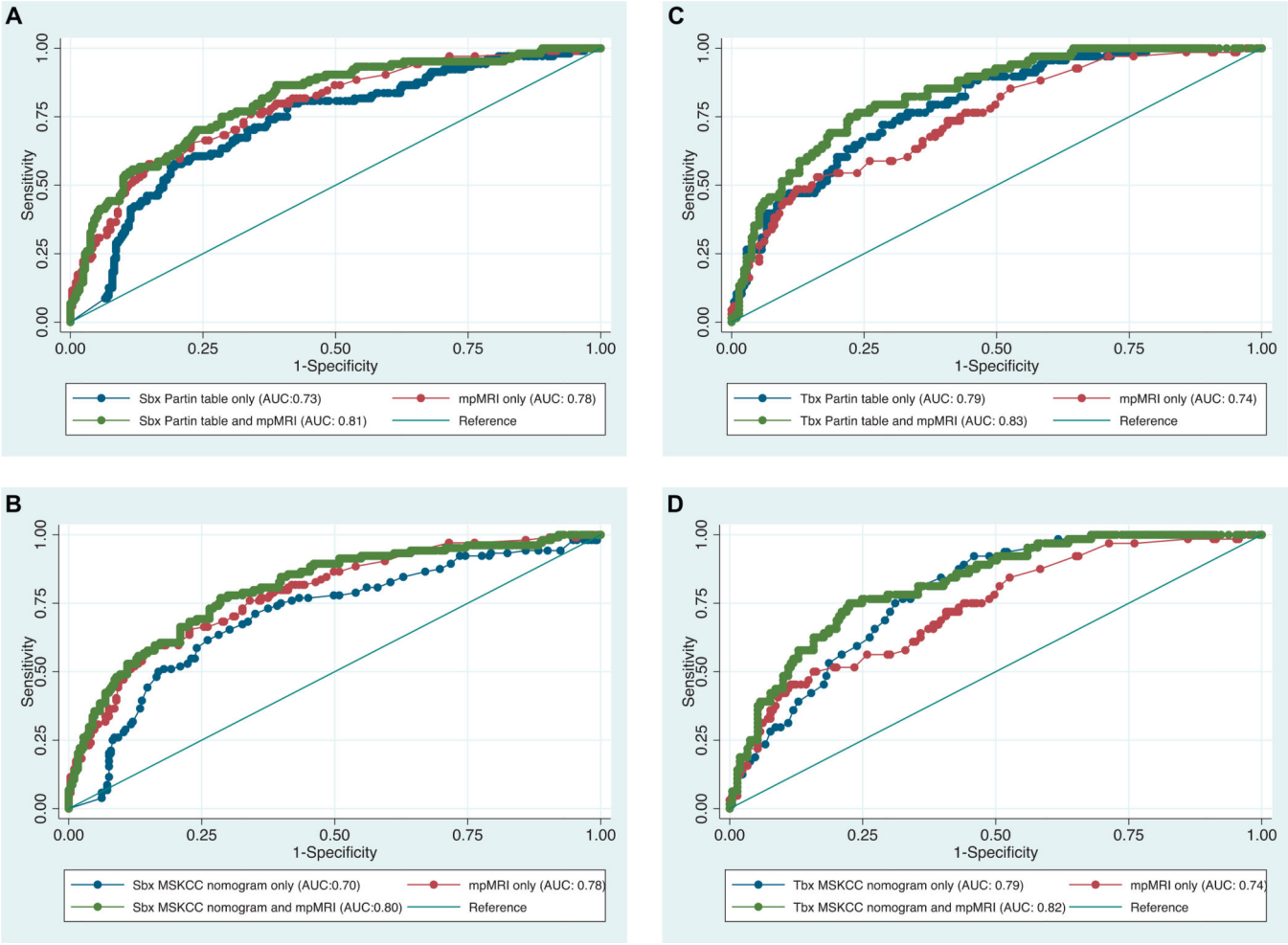

Figure.

ROC curves of Partin[3] and MSKCC[4] nomograms by biopsy type with and without adding mpMRI results to predict nonorgan confined disease. *A*, SBx and Partin nomogram. *B*, Sbx and MSKCC nomogram. *C*, Tbx and Partin nomogram. *D*, Tbx and MSKCC nomogram.

Table 1.

Preoperative clinical variables

Median age (IQR)	61	(56–66)
No. clinical stage (%): *		
cT1	483	(90.8)
cT2	49	(9.2)
Median ng/ml PSA (IQR)	6.2 (4.3–9.9)	
No. systematic biopsy Grade Group (%):		
1	190	(36.0)
2	201	(37.8)
3	46	(8.6)
4	73	(13.8)
5	22	(3.8)
No. targeted biopsy Grade Group (%):		
1	82	(25.1)
2	112	(34.3)
3	41	(12.5)
4	76	(23.2)
5	16	(4.9)
mpMRI findings: †		
No. low NIH suspicion score (%)	52	(9.8)
No. intermediate NIH suspicion score (%)	254	(47.7)
No. high NIH suspicion score (%)	226	(42.5)
No. suspected extraprostatic extension (%)	101	(19.0)
No. suspected seminal vesicle invasion (%)	24	(4.5)
Mean ± SD largest lesion diameter (cm)	1.6 ± 0.7	

*No cT3 disease.

†No suspected lymph node invasion.

Table 2.

ROC AUCs of MSKCC[4] and Partin[3] nomograms with and without adding mpMRI results

Nomogram	mpMRI Model AUC (95% CI)	Systematic TRUS Biopsy			Targeted TRUS Biopsy		
		Nomogram AUC (95% CI)	Nomogram + mpMRI AUC (95% CI)	p Value	Nomogram AUC (95% CI)	Nomogram + mpMRI AUC (95% CI)	p Value
MSKCC:							
Organ confined disease	0.78 (0.72e0.83)	0.70 (0.64e0.76)	0.80 (0.75e0.85)	<0.001 *	0.77 (0.73e0.84)	0.82 (0.76e0.87)	0.046 *
Extraprostatic extension	0.78 (0.71e0.82)	0.70 (0.63e0.77)	0.80 (0.74e0.85)	0.003 *	0.77 (0.73e0.83)	0.82 (0.75e0.87)	0.045 *
Seminal vesicle invasion	0.86 (0.75e0.92)	0.81 (0.71e0.88)	0.90 (0.83e0.96)	0.011 *	0.76 (0.67e0.86)	0.85 (0.72e0.93)	0.065
Lymph node invasion	0.87 (0.77e0.92)	0.82 (0.70e0.89)	0.88 (0.83e0.94)	0.120	0.88 (0.84e0.95)	0.94 (0.91e0.97)	0.023 *
Partin:							
Organ confined disease	0.78 (0.72e0.83)	0.73 (0.67e0.78)	0.81 (0.75e0.86)	0.001 *	0.78 (0.74e0.84)	0.83 (0.76e0.88)	0.030 *
Extraprostatic extension	0.78 (0.71e0.82)	0.66 (0.59e0.71)	0.80 (0.73e0.83)	<0.001 *	0.67 (0.61e0.75)	0.79 (0.73e0.85)	0.003 *
Seminal vesicle invasion	0.86 (0.75e0.92)	0.80 (0.71e0.89)	0.88 (0.82e0.96)	0.022 *	0.83 (0.69e0.88)	0.87 (0.77e0.96)	0.452
Lymph node invasion	0.87 (0.77e0.92)	0.85 (0.68e0.88)	0.91 (0.85e0.96)	0.0710	0.89 (0.84e0.97)	0.93 (0.86e0.97)	0.085

* Significant at $p < 0.05$.