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Review Article

Prostate cancer nomograms and their application in Asian men: a review

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ABSTRACT

Nomograms help to predict outcomes in individual patients rather than whole populations and are an important part of evaluation and treatment decision making. Various nomograms have been developed in malignancies to predict and prognosticate clinical outcomes such as severity of disease, overall survival, and recurrence-free survival. In prostate cancer, nomograms were developed for determining need for biopsy, disease course, need for adjuvant therapy, and outcomes. Most of these predictive nomograms were based on Caucasian populations. Prostate cancer is significantly affected by race, and Asian men have a significantly different racial and genetic susceptibility compared to Caucasians, raising the concern in generalizability of these nomograms. We reviewed the existing literature for nomograms in prostate cancer and their application in Asian men. There are very few studies that have evaluated the applicability and validity of the existing nomograms in these men. Most have found significant differences in the performance in this population. Thus, more studies evaluating the existing nomograms in Asian men or suggesting modifications for this population are required.

1. Introduction

Prostate cancer (PCa) is the second most common cancer diagnosed in men and fifth most common oncologic cause of mortality among men. As per GLOBOCON 2020 data, approximately 1.4 million men were diagnosed with PCa worldwide.[1] The incidence of PCa is higher in Western countries than in Eastern and South Central Asia.[2] Mortality rates of PCa vary worldwide and high rates are found in African decent populations and very low rates in Asia.[3] PCa is a disease of older people with a median age of 68 years. It has been estimated that in Europe and the United States, the diagnosis of PCa in men over 65 years of age will cause a 70% increase in annual diagnosis by 2030.[4,5] Men with intermediate- and high-risk PCa benefit the most from active treatment while advanced age and poor performance status decreases the benefit of intervention with curative treatment.[6]

Nomogram are predictive tools for clinical outcomes based on a set of variables. They assist in making predictions for individual patients rather than for population risk groups and are thus more applicable while assessing a single patient. Nomograms aid in risk assessment and decision making by predicting outcomes with different treatment modalities. PCa is a diagnosis particularly suited to the use of nomograms since there are a multitude of treatment options with extremely varying outcomes and nomograms have become an essential part of decision making in these men.

A number of nomograms are available for PCa. In men with clinically localized PCa, the Partin tables[7] were among the first and most widely used nomograms for patient counseling. Other nomograms include those for decision on active surveillance,[8] radical prostatectomy (RP),[9] neurovascular bundle preservation,[10] exclusion of pelvic lymph node dissection during RP,[11] brachytherapy,[12] external beam radiation therapy,[13] prediction of biochemical recurrence (BCR) free survival,[14] outcomes of adjuvant radiotherapy (RT),[15] prediction of metastasis,[16] and cancer specific mortality.[17]

In PCa, race is well known to affect disease outcomes,[18] and it has been documented that prostate specific antigen (PSA), one of the most common variables in PCa nomograms, is a poor predictor of disease in Indian men.[19] Due to racial differences in Asian and Western populations, detection of PCa varies with PSA levels.[20,21] PCa detection rates may be only 15–26% in Asian men with PSA between 4 and 10 ng/ml.[20,22] The PSA values may vary even in the same individual and merit a retest before a biopsy.[23]

The prevalence of PCa is higher in western countries and underlying gene susceptibility, positive family history, racial and

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clinical differences, disease aggressiveness, high-risk disease stages have a role in developing the risk calculators and nomograms. Vidal et al looked at the data from the Reduction by Dutasteride of Cancer Events study and found Asian men to have a lower risk of PCa diagnosis and suggested that this could be due to biological, genetic, or lifestyle factors.[24] A higher incidence of acute inflammation in the prostate biopsy in Asian men may also have a role to play in the difference on cancer diagnosis.[25] Asian men have been shown to have lower 5α-reductase activity, and it has been proposed that this genetic variation may be responsible for their lower risk of PCa.[26] Miyake et al suggest that there may be a role of gut and urinary microbiome in causation of PCa and it remains to be seen if this could also be a cause for genetic variations.[27] Thus, nomograms developed in the western populations where disease prevalence is much more than in Asia may have limited predictive power in Asian populations.[28] For this review, we evaluated PCa nomograms for their applicability in Asian men.

2. Material and methods

The literature search was performed through PubMed/Medline, Embase, Web of Science, and Google Scholar databases. The following MeSH keywords were used to search all relevant papers in English literature (nomograms OR predictive nomograms) AND (PCa OR prostate acinar adenocarcinoma OR prostate malignancy OR prostate neoplasm). Abstracts, full articles including systematic review and meta-analyses, were reviewed for the relevant contents. Case reports, letters, brief communications, editorials, and articles in non-English language were excluded.

3. Results

3.1. Pre-biopsy nomograms

Prostate biopsy is an invasive procedure with associated complications. All men with a clinical suspicion of PCa do not have cancer and a biopsy may be negative even in men with cancer. Thus, the decision to perform a biopsy can often not be based on clinical suspicion alone or a single variable and nomograms can help predict the yield of a biopsy and thus decide on whether a biopsy should be obtained. This becomes particularly relevant when all PCa is not aggressive and detection of indolent cancer adds to anxiety with little impact on survival.[29] The common pre-biopsy risk calculators that help to predict the detection of clinically significant cancer on a prostate biopsy are the European Randomized Study of Screening for Prostate Cancer (ERSPC) risk calculator, Prostate Cancer Prevention Trial (PCPT) risk calculator, and the Sunnybrook nomogram.[30,31]

Chun et al developed and validated a nomogram on extended biopsy sampling and contemporary repeat biopsy nomogram.[32,33] Zhu et al compared these nomograms and found superior predictive assessment of ERSPC risk calculator compared to PCPT risk calculator in a Chinese cohort.[34] Similarly, Yoon et al validated the ERSPC risk calculator in the Korean cohort.[35] Both studies noted that the biopsy nomograms significantly overestimated the risk in Chinese and Korean cohorts compared to the western populations. Table 1 describes some Asian PCa risk prediction models showing the PCa detection rate.

In recent years, multiparametric magnetic resonance imaging (MRI) has shown a promising role as a pre-biopsy tool and multiparametric MRI-based nomograms by Van Leeuwen et al,[36] Bjurlin et al,[37] and Radtke et al, [38] showed high discrimination for predicting clinically significant PCa.

3.2. Pre-treatment nomograms

Adverse pathological features on final histopathology, such as seminal vesicle invasion, extracapsular disease, and lymph node positivity, may necessitate early adjuvant RT in post-RP. Hence, models that can predict adverse pathological stage at pre-prostatectomy setting can be helpful in counseling men undergoing RP.

Various pre-prostatectomy nomograms (Table 2) are available to determine pathological stage, clinically indolent cancer, organ confined disease, surgical margin positivity, capsular penetration, Gleason grade upgrading between biopsy and RP, seminal vesical invasion, lymph nodal positivity, and extracapsular extension risk before RP.

The most common variables used in these prediction models were PSA, Gleason sum, and clinical stage of PCa. Further, Steuber et al [39] described a nomogram that predicts tumor location (peripheral zone vs. transitional zone) taking number of positive biopsy cores at the base and mid gland level along with cumulative percent biopsy tumor volume. Likewise, side-specific percent positive cores and tumor volume at base, mid, and apex were included in nomograms predicting side-specific extracapsular extension and organ confined disease, respectively.

Kattan's nomogram was among the first pre-treatment nomograms developed to predict the 5-year BCR in men undergoing RP with an external validation accuracy of 65–83%.[40] Though promising, Kattan's model was limited by a smaller follow-up period of 5 years. Stephenson et al later addressed this by a 10-year predictive model on biochemical recurrence with 76–79% discrimination.[41] Table 3 lists various models developed in the pre-treatment prediction of BCR in men treated with RT.

3.3. Post-prostatectomy nomograms

BCR and survival in PCa depend on the pathological stage of the disease and a number of models have been developed to predict

Table 1
Asian prostate cancer risk prediction models

Study	Population	Variables	PCa detection rate (%)
Suzuki et al[54]	Japanese	Age, PSA, PV, DRE, % fPSA	28.9
Park et al[55]	Korean	Age, PSA, DRE, Prostate TZ volume	28.6
Yoon et al[35]	Korean	Age, PSA, DRE, Prostate TZ volume	28.6
Tang et al[28]	Chinese	Age, PSA, PV, DRE	44.8
Kuo et al[56]	Chinese	Age, PSA, PV, DRE, TRUS echogenicity	34.4
Jeong et al[49]	Korean	Age, PSA, PV, TRUS, DRE	35.6
Huang et al[57]	Chinese	Age, PSA, PV, DRE, %fPSA, TRUS	41.5
Wu et al[58]	Chinese	Age, PSA, PV, DRE, %fPSA, TRUS	45.3
Chen et al[21]	Chinese	Age, PSA, PV, DRE, %fPSA,	36.6

PCa-prostate cancer; PSA-prostate-specific antigen; PV- prostate volume; DRE-digital rectal examination; %fPSA-percentage free PSA; TZ-transitional zone; TRUS- transrectal ultrasonogram.

Table 2
Nomograms on prediction of pathologic stage in men treated with radical prostatectomy for clinically localized prostate cancer

Study	Prediction model	Outcome measure	No of Patients	Variables	Discrimination
Partin et al[59]	Probability table	Pathologic stage	703/4133	Biopsy GS, CS, PSA	Internal: 72% External: 84%
Epstein et al[7]	Risk group	Clinically indolent cancer defined as pathologically organ confined, tumor volume $\leq$0.2 cc, GS < 7	157	Biopsy GS, millimeter core with cancer, PSAD, no adverse pathologic findings on needle biopsy	NA
Kattan et al[8]	Probability nomogram development	Clinically indolent cancer defined as pathologically organ confined, tumor volume $\leq$0.5 cc, no GG 4 or 5	409	PSA, primary and secondary biopsy GS, volume, millimeter core with cancer, millimeter core without cancer	64%
Chun et al[60]	Probability nomogram development	Gleason upgrading between biopsy and RP	2982	PSA, CS, primary and secondary Biopsy GS	80%
Chun et al[61]	Probability nomogram development	Significant Gleason upgrading between biopsy and RP	4789	PSA, CS, biopsy GS	76%
Ackerman et al[62]	Probability formula	Surgical margin positivity	107	Number positive sextant cores, PSAD	70%
Bostwick et al[63]	Probability graph	Capsular penetration	314	Biopsy GS, Percent cancer in biopsy cores, PSA	78%
Gamito et al[64]	Neural network	Capsular penetration	4133	Age, race, PSA, PSAV, GS, CS	30–76%
Gilliland et al[65]	Probability graph	ECE	3826	Age, biopsy GS, PSA	63%
Steuber et al[39]	Probability nomogram development	Side-specific ECE	1118	PSA, CS, biopsy GS, percent Positive cores, percent of cancer in positive cores	84%
Baccala et al[66]	Probability nomogram development	SV invasion	6740	Age, PSA, Biopsy GS, CS	80%
Gallina et al[67]	Probability nomogram development	SV invasion	896	PSA, CS, biopsy GS, percent positive biopsy cores	79%
Bluestein et al[68]	Probability graph	LN invasion assessed with limited pelvic lymphadenectomy	816	Biopsy GS, CS, PSA	82%
Batuello et al[69]	Neural network	LN invasion assessed with limited pelvic lymphadenectomy	6454	Biopsy GS, CS, PSA	77–81%
Briganti et al[70]	Probability nomogram development	LN assessed with extended pelvic lymphadenectomy ($\geq$10 nodes)	602	PSA, CS, biopsy GS	76%
Kim et al[50]	Probability nomogram development	LN invasion assessed with extended pelvic lymphadenectomy	541	PSA, CS, GS	Internal: 88.3% External: NA

GS, Gleason sum; CS, clinical stage; PSA, prostate specific antigen; PSAD, prostate specific antigen density; GG, Gleason grade; RP, radical prostatectomy; PSAV, prostate specific antigen velocity; ECE, extracapsular extension; SV, seminal vesicle; LN, lymph node; NA, not available.

Table 3
Nomograms on pre-treatment prediction of biochemical recurrence in men treated with radiotherapy

Study	Prediction model	BCR (years)	Radiation type	No of patients	Variables	Discrimination
Zagars et al[71]	Probability graph	6	EBRT	938	PSA, biopsy GS, CS	NA
D'Amico et al[72]	Probability table	2	EBRT	762	Biopsy GS, CS, PSA	NA
Shipley et al[73]	Probability table	5	EBRT	1607	Biopsy GS, CS, PSA	NA
Kattan et al[74]	Probability nomogram development	5	EBRT	1042/1030	PSA, biopsy GS, CS, neoadjuvant ADT, radiation dose delivered	73%
D'Amico et al[75]	Probability graph	5	EBRT	766	Biopsy GS, CS, PSA, treatment modality	NA
Ragde et al[76]	Risk group	10	BT	98	Age, biopsy GS, CS, PSA, 45 Gy EBRT	76%
Kattan et al[12]	Probability nomogram development	5	BT	920, 1827, 765	Biopsy GS, CS, PSA, co-administration of EBRT	61–64%

BCR, biochemical recurrence; EBRT, external beam radiotherapy; BT, brachytherapy; GS, Gleason sum; ADT, androgen deprivation therapy; NA, not available.

Table 4
Pre- and post-operative prediction of biochemical recurrence in men treated with radical prostatectomy

Study	Prediction model	BCR years	Number of patients	Variables	Discrimination
Kattan et al[40]	Probability nomogram development	5	983	Biopsy primary and secondary GG, CS, PSA	Internal: 74% External: 65–83%
D'Amico et al[72]	Probability table	2	892	Biopsy GS, CS, PSA	NA
D'Amico et al[77]	Probability graph	2	977	Biopsy GS, endorectal coil MRI T-stage, PSA, percent positive biopsy cores	NA
Tewari et al[78]	Neural network	3.5	1400	Age, race, PSA, CS, biopsy GS	83%
D'Amico et al[79]	Probability graph	4	823	Biopsy GS, CS, PSA, percent positive biopsy cores	80%
Cooperberg et al[80]	Probability graph	3 and 5	1439	Age, PSA, biopsy GS, CS, percent positive biopsy	Internal: 66% External: 68–81%
Stephenson et al[41]	Probability nomogram development	10	1978,1545	PSA, CS, biopsy GS, year of surgery, number of positive and negative cores	76–79%
D'Amico et al[79]	Probability graph	2	862	Pathologic stage, PSA, GS, surgical margin status	NA
Kattan et al[42]	Probability nomogram development	5	996	PSA, GS, ECE, SV invasion, LN invasion, surgical margin status	Internal:89% External: 77–83%
McAleer et al[81]	Probability graph	7	2417	GG, CS, margin status, dichotomized PSA (cut point 10 ng/mL)	NA
Stephenson et al[43]	Probability nomogram development	10	1881, 1782, 1357	PSA, GS, ECE, SV invasion, LN invasion, surgical margin status	78–86%
Suardi et al[44]	Probability nomogram development	5, 10, 15, and 20	601, 2963, 3178	GS, pathologic stage, surgical margin status, type of surgery, adjuvant RT	Internal: 77–81% External: 77–86%

BCR, biochemical recurrence; GS, Gleason sum; CS, clinical stage; GG, Gleason grade; RP, radical prostatectomy; MRI, magnetic Resonance imaging; ECE, extracapsular extension; SV, seminal vesicle; LN, lymph node; RT, radiotherapy; NA, not available.

Table 5
Nomograms on prediction of metastasis and survival

Study	Prediction model	Patient population	Outcome measure	No of Patients	Variables	Discrimination
Partin et al[82]	Probability graph	RP	Local versus distant recurrence	1058	PSAV, GS, pathologic stage	NA
Pound et al[83]	Probability table	BCR after RP	Metastasis (7 years)	315	PSADT, GS, time to BCR	56%
D'Amico et al[84]	Probability graph	RP	PCa-specific mortality (8 years)	4946	Biopsy Gleason sum, CS, PSA	NA
Freedland et al[85]	Probability table	BCR after RP	Cancer-specific survival (10 years)	379	PSADT, GS, time from surgery to BCR	59%
D'Amico et al[84]	Probability graph	EBRT	PCa-specific mortality (8 years)	2370	Biopsy GS, CS, PSA	NA
Kattan et al[86]	Probability nomogram development	EBRT	Metastasis (5 years)	1677, 1626	PSA, CS, biopsy GS	81%
Zhou et al[87]	Probability graph	EBRT	PCa-specific mortality (5 years)	661	PSADT, biopsy GS	NA
Stephenson et al[88]	Probability nomogram development	Salvage RT for BCR after RP	BCR after RT (7 years)	1540	Prostatectomy PSA, GS, SV invasion, ECE, surgical margin status, LN metastasis, persistently elevated PSA after RP, pre-radiotherapy PSA, PSADT, neoadjuvant ADT, radiation dose	69%
Zhou et al[87]	Probability graph	BCR after RP	PCa-specific mortality (5 years)	498	PSADT	NA
Slovin et al[16]	Probability nomogram development	BCR after RP or RT	Metastasis (1–2 years)	148	Baseline PSA, PSADT, Pathologic T stage, GS	69%
Smaletz et al[89]	Probability nomogram development	progressive metastatic PCa after castration	OS (1–2 years)	409, 433	Age, Karnofsky performance index, hemoglobin, PSA, lactic dehydrogenase, alkaline phosphatase, albumin	71%
Halabi et al[90]	Probability nomogram development	Metastatic HRPC	OS (1–2 years)	1101	Lactate dehydrogenase, PSA, alkaline phosphatase, GS, Eastern Cooperative Oncology Group performance status, hemoglobin, presence of visceral disease	68%

RP, radical prostatectomy; PSA, prostate specific antigen; PSADT, prostate specific antigen doubling time; BCR, biochemical recurrence; PCa, prostate cancer; OS, overall survival; GS, Gleason sum; CS, clinical stage; GG, Gleason grade; ECE,extracapsular extension; SV, seminal vesicle; LN, lymph node; EBRT, external beam radiotherapy; RT, radiotherapy; HRPC, hormone-refractory prostate cancer; ADT, androgen deprivation therapy; NA, not available.

Table 6
Nomograms on prediction of life expectancy in men with clinically localized prostate cancer

Study	Prediction model	Outcome measure	No of Patients	Variables	Discrimination
Albertson et al[91]	Probability formula	OS (10 years)	451	Age, GS and index of coexistent disease category	71%
Tewari et al[92]	Probability graph	OS (10 years)	6149	Age, race, comorbidity, PSA, GS, treatment type	63%
Cowen et al[93]	Probability nomogram development	Life expectancy (5–15 years)	506	Age, CCI, presence of angina, systolic blood pressure, body mass index, smoking, marital status, PSA, GS, CS, treatment type	73%
Walz et al[45]	Probability nomogram development	Life expectancy (10 years)	9131	Age, CCI, treatment type	84.3%

OS, overall survival; CCI, Charlson comorbidity index; RP, radical prostatectomy; PSA, prostate specific antigen; PSADT, prostate specific antigen doubling time; BCR, biochemical recurrence; GS, Gleason sum; CS, clinical stage; EBRT, external beam radiotherapy.

BCR after RP (Table 4). The post-prostatectomy model by Kattan et al estimated 5-year BCR in men who underwent RP for localized PCa.[42] The model's accuracy in external validation ranged between 77 and 83%. In contrast, Stephenson's 10-year post-prostatectomy model for BCR yielded a discrimination of 78-86% on external validation.[43] Suardi et al developed the furthest-reaching 20-year BCR prediction tool in the post-prostatectomy setting.[44] Their prediction model had Gleason sum, pathologic stage, surgical margin status, type of surgery, and adjuvant RT as variables. The model's discrimination ranged between 77 and 86% confirmed in two external validation cohorts. Table 5 describes various models in predicting metastasis and PCa-specific mortality after definitive treatment.

3.4. Nomograms predicting life expectancy

In PCa, life expectancy is an important factor for informed decision making in men eligible for definitive treatment. In general, 10 years of life expectancy is accepted as the minimum prerequisite for treatment with curative intent. Table 6 describes nomograms on the prediction of life expectancy in men with clinically localized PCa. Walz et al developed a life expectancy model with age and comorbidities as variables in men undergoing definitive treatment. This tool has a higher discrimination of 84.3%.[45] In contrast, the MALE predictive model required detailed information on specific cardiac comorbidities in predicting life expectancy.[46] While there are many nomograms for life expectancy, however, their clinical application is less. Kim et al. estimated that only 25% of radiation oncologists or urologists use life expectancy.[47]

3.5. Predictive nomograms incorporating MRI

In recent years, the role of MRI in PCa has increased with improvement in the technology. Incorporation of MRI variables with other clinical variables increased the accuracy in predictive nomograms. Wang *et al.* investigated the value of endorectal coil MRI with magnetic resonance spectroscopic imaging to the staging nomograms for predicting organ-confined PCa. The model predicted seminal vesicle invasion with a discriminatory accuracy of 87%.[48] Likewise, incorporation of MRI variables such as the presence of extracapsular extension or seminal vesicle invasion had a better discrimination (Area under curve [AUC] = 89% vs. 63%, $p < 0.01$) in predicting lymph node invasion than the base Partin model.[48]

4. Discussion

Western nomograms are mainly developed based on the screening cohort compared to Asian nomograms which are mainly based on clinical cohort. Family history is an important variable in western nomograms compared to Asian nomogram due to the high incidence of positive family history in western populations.

In the Asian context, the Seoul National University Prostate Cancer Risk Calculator was developed based on data from 3482 Korean men who underwent prostate biopsies. They also showed that in the validation cohort of 1112 Korean men, the AUC of Seoul National University Prostate Cancer Risk Calculator outperformed ERSPC and PCPT risk calculators (AUC 0.811 vs. 0.768 vs. 0.704, respectively).[49]

Similarly, the Chinese Prostate Cancer Consortium Risk Calculator by Chen et al. was based on age, logPSA, logPV, free PSA ratio, and digital rectal examination variables in their model and showed that Chinese Prostate Cancer Consortium Risk Calculator had a better discrimination and calibration and decision curve analysis in such population as compared with ERSPC and PCPT risk calculators.[21]

Kim et al. developed a model in predicting LN invasion in Asian men undergoing RARP with pelvic LN dissection for localized PCa. The bootstrapped corrected AUC of this model with PSA, clinical stage, and biopsy Gleason sum as variables was 0.883. Further, with a cut off value of 4%, the authors showed they could omit pelvic lymph node dissection in 60.2%, missing only two patients (4.4%) with LN invasion.[50]

A recent study from the Indian subcontinent showed that incorporating MRI extracapsular extension risk score to the clinicopathological variables in Partin nomogram had an incremental value in predicting extracapsular extension in men undergoing RP. Their model had a higher predictive accuracy than the Partin nomogram (AUC 0.82 vs. 0.67, $P < 0.00023$).[51]

In terms of functional and oncological outcomes, Sharma et al developed preoperative and postoperative nomograms predicting quadrifecta following Robot-assisted radical prostatectomy (RARP) in Indian men. Both models were internally validated, and on Receiver operating characteristic (ROC) analysis, preoperative and postoperative nomograms had an area under the curve of 71 and 79%, respectively.[52]

In a Japanese cohort, Blas et al developed a novel nomogram predicting biochemical recurrence-free survival following RP by including pathological stage and Gleason sum, positive surgical margin, PSA $\geq$0.05 ng/mL at one year and LN metastasis as variables. On comparing their model with the United States-based Cancer of the Prostate Risk Assessment post-Surgical score using the same validation cohort, the authors showed a higher c-index (0.89 vs. 0.78, $P = 0.01$) and a positive net benefit at 3 and 5 years postoperatively in the decision curve analyses.[53]

To be generalizable, a nomogram should be accurate in population other than the original cohort from which they developed the model. However, in PCa, there is substantial variation in the nomograms with varied discriminatory accuracy. Further, the availability of such tools in the non-Caucasian cohort is limited. Hence, a separate nomogram may be needed for non-Caucasian men with different racial and clinical profiles and also a low susceptibility of high-risk disease. Further, inclusion of image based or molecular marker in nomograms may be needed to predict distinct clinical outcomes in Asian men with PCa.

5. Conclusion

Nomograms help to individualize predictive outcomes and help patients with PCa to make an informed decision based on their outcome and risk prediction. Most predictive models are based on Caucasian populations with only a few models available for non-Caucasian populations, affecting their generalizability. Studies evaluating their validity in Asian men are required enable better prediction of outcomes in Asian men.

Conflicts of interest

The authors have no conflicts of interest.

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