PATHOLOGY

Updated Nomogram to Predict Pathologic Stage of Prostate Cancer Given Prostate-Specific Antigen Level, Clinical Stage, and Biopsy Gleason Score (Partin Tables) Based on Cases from 2000 to 2005

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Objectives: To update the 2001 "Partin tables" with a contemporary patient cohort and revised variable categorization, correcting for the effects of stage migration.
Methods: We analyzed 5730 men treated with prostatectomy (without neoadjuvant therapy) between 2000 and 2005 at the Johns Hopkins Hospital. Average age was 57 years. Multivariable logistic regression was used to estimate the probability of organ-confined disease, extraprostatic extension, seminal vesicle involvement, or lymph node involvement. Predictor variables included preoperative prostate-specific antigen (PSA) level (0 to 2.5, 2.6 to 4.0, 4.1 to 6.0, 6.1 to 10.0, and greater than 10.0 ng/mL), clinical stage (T1c, T2a, and T2b/T2c), and biopsy Gleason score (5 to 6, 3 + 4 = 7, 4 + 3 = 7, or 8 to 10). Bootstrap resampling was used to generate 95% confidence intervals for predicted probabilities.
Results: Seventy-seven percent of patients had T1c, 76% had Gleason score 5 to 6, 80% had a PSA level between 2.5 and 10.0 ng/mL, and 73% had organ-confined disease. Nomograms were developed for the predicted probability of pathologically organ-confined disease, extraprostatic extension, seminal vesicle invasion, or lymph node involvement. The risk of non-organ-confined disease increased with increases in any individual prognostic factor. The dramatic decrease in clinical stage T2c compared with the patient series used in the previous models resulted in T2b and T2c being combined as a single predictor in the nomogram.
Conclusions: These updated "Partin tables" were generated to reflect trends in presentation and pathologic stage for men diagnosed with clinically localized prostate cancer at our institution. Clinicians and patients can use these nomograms to help make important decisions regarding management of prostate cancer.

Editorial Comment

It is worth noting in this updated nomogram that Gleason score 7 has been stratified to 3+4=7 and 4+3=7. Tumors with a Gleason score of 7 have a significantly worse prognosis than those with a Gleason score of 6. Given the adverse prognosis associated with Gleason pattern 4, one would expect that whether a tumor is Gleason score 3+4 or 4+3 would influence prognosis (1). This issue has been controversial in the literature, however, most of the studies have shown that Gleason score 4+3 has a worse prognosis than Gleason score 3+4 (2,3). Recently we evaluated the biochemical (PSA) progression following radical prostatectomy in 300 patients according to Gleason score 3+4 and 4+3 in the surgical specimens. Of the total of 300 patients, 140/300 (46.6%) patients were Gleason score 3+4=7 and 37/300 (12.3%) patients Gleason score 4+3=7. The 4-year biochemical (PSA) progression-free survival rate with Gleason score 3+4 and Gleason score 4+3 was 60% and 30%, respectively (log-rank, p=0.046).

Another topic in the updated nomogram relates to clinical stage. According to the authors the dramatic decrease in clinical stage T2c compared with the present series used in the previous models resulted in T2b and T2c being combined as a single predictor in the nomogram. According to the 2002 TNM classification of malignant tumors, T2b involves more than half of one lobe, but not both lobes. Some studies did not found this stage in surgical specimens. Eichelberger & Cheng (4) question the existence of a true pathologic stage pT2b tumor. They studied 369 prostate cancer patients treated by radical prostatectomy. Prostate cancers were multifocal in

312 cases (85%). The majority of the specimens were pathologic stage pT2 (276, or 75%). Using the 2002 TNM staging criteria, 54 (15%) of the tumors were stage pT2a, 222 (60%) were pT2c, 75 (20%) were pT3a, and 18 (5%) were pT3b. No pathologic stage pT2b tumors were identified. The findings of Quintal et al. (5) using a point-count method for evaluating tumor extension, are in accordance with Eichelberger and Cheng (3). No tumor pathologic stage pT2b was found and the frequency of the stages in Quintal's series is very similar to theirs: stage pT2a, 28 (12.50%); pT2c, 138 (61.61%); pT3a, 30 (13.39%); and, pT3b 28 (12.50%).

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Basal Cell Carcinoma of the Prostate: A Clinicopathologic Study of 29 Cases

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We studied 29 cases of basal cell carcinoma of the prostate including what others call adenoid cystic carcinoma of the prostate. Patients' age ranged from 42 to 89 (mean 69) years. The most common methods of diagnosis was transurethral resection (TURP) (n=29) and needle biopsy (n=9). In 28/29 cases, slides were reviewed and 24 (86%) cases showed more than 1 pattern: adenoid cysticlike (AC-P) pattern and small solid nests with peripheral palisading were the most predominant patterns, each seen in 18 cases (64%). Other patterns included: basal cell hyperplasialike in 9 cases (32%); small tubules occasionally lined by a hyaline rim in 9 cases (32%), with 4 of these cases also demonstrating intermingling cords of cells; and large solid nests in 8 cases (28.5%), 5 of which had central necrosis. Fourteen cases of small nests and tubules were centrally lined by eosinophilic cells. Desmoplasia was noted in 20 (71%) cases. Infiltration around benign glands was seen in 10 (36%) cases, with predominantly small nests and AC-P. Invasion of thick muscle bundles of the bladder neck was seen in 10 of 21 TURP cases. Perineural invasion was noted in 3 cases with AC-P and 1 case of small basaloid nests. Perineural and vascular invasion was seen in 2 basal cell carcinomas with large basaloid nests.

Mitoses ranged from 0 to 60/10 hpf (mean=4). bcl2 was diffusely positive in 22/24 (92%) cases. Ki67 ranged from 2% to 80% (mean=23%). Ki67 > or =20% was seen in 13 (56.5%) cases, including all patterns except small solid nests. Basal cell markers (HMWCK, p63) either: (1) highlighted multiple layers of cells in 15/25 (60%) cases with sparing of the inner most luminal layer; (2) labeled just the outermost layers in 6/25 (24%) cases; or (3) reacted with only a few scattered cells in 4/25 (16%) cases (3 with large solid nests with central necrosis, 1 with tubules and cords). Seven patients had RP with: 5/7 showing extraprostatic extension with 1/5 also showing seminal vesicle involvement and 2/5 also with a positive margin; 1/7 having organ confined disease; and 1/7 showing no residual disease. An additional 11 cases showed extraprostatic extension on TURP with bladder neck invasion (n=10) or periprostatic adipose tissue invasion (n=1). Of 29 (65.5%) cases, 19 had follow-up > 1 year with a mean of 4.3 years (1 to 19 y). Of 19 (77%) cases, 14 had no evidence of disease after 1 to 19 (mean 5.8) years. Of 19 patients, 4 locally recurred with 2 after TURP, 1 after enucleation, and 1 after RP. Metastases developed in 4/29 patients: 1 in lung, 1 in lung and liver, 1 in lung, bone and liver, 1 in penile urethra. Basal cell carcinomas are rare tumors with a broad morphologic spectrum. These tumors predominantly show an indolent course with local infiltrative behavior. A small subset behaves aggressively with local recurrences and distant metastases. The most common morphology among those with an aggressive behavior is large solid nests more often with central necrosis, high Ki67%, and less staining with basal cell markers.

Editorial Comment

Basal and stem cells comprise the proliferative compartment of the prostatic acinus. There is a spectrum of basal cell lesions including typical hyperplasia, atypical hyperplasia, adenoma, and carcinoma (or adenoid cystic carcinoma). The latter is a rare tumor initially considered with an indolent biologic potential (1). In 2003, Iczkowski et al. (2) published the largest series at that time calling attention to the potential aggressiveness of this tumor requiring ablative therapy. From a total of 19 patients, 54 (21%) developed metastases.

Ali and Epstein's is the largest series so far of basal cell carcinoma (or adenoid cystic carcinoma) of the prostate. Of a total of 29, 19 patients had follow-up >1 year: 14 patients had no evidence of disease after 1 to 19 (mean 5.8 years); 4 locally recurred and 4 developed metastases.

The authors conclude that these tumors predominantly show an indolent course with local infiltrative behavior. A small subset behaves aggressively with local and distant metastases. The most common morphology among those with aggressive behavior is large solid nests more often central necrosis, high Ki67%, and less staining with basal cell markers.

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