

were grouped into TP+ve cancers (detected through TP14PBx) and TP-ve cancers (not detected through TP14PBx but detected through TR12PBx). The clinicopathological characteristics of the two groups were evaluated. For analysis of cancer location, the prostate was divided into the apex, midprostate, and base. The apex/base was defined as the most inferior/superior 10 mm of the gland, respectively. The remaining part of the gland was defined as the midprostate. When a significant cancer focus lay astride two regions, it was assigned to both regions.

RESULTS: Median age and PSA were 66 years and 6.1 ng/mL, respectively. TP-ve cancers comprised 14% of all cancers. Biopsy Gleason scores (GS) of TP-ve and TP+ve cancers were 5-6/3+4/4+3/8-10 in 46/24/16/14% and in 30/27/17/26%, respectively (no significant difference). The median number of positive cores in TP-ve cancers was significantly smaller than that in TP+ve cancers (1 vs. 5, $p < 0.001$). In RP specimens, the rate of index tumor volume ≥ 0.5 mL was significantly smaller in TP-ve cancer than in TP+ve cancer (26% vs. 84%, $p < 0.001$), but the distribution of RP GS had no significant difference between the two groups. The location of TP-ve and TP+ve cancers was 85/65/20% and 96/93/29% in the apex/midprostate/base, respectively (no significant difference).

CONCLUSIONS: In an initial setting, only 14% of cancers detected by 3D26PBx were missed by TP14PBx, and many of the TP14PBx-missed cancers were low-volume cancers. Most of the cancers missed by TP14PBx were located in the apex of the prostate, with a few in the base.

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2214 ASSOCIATION BETWEEN PROSTATE CALCIFICATION SUBTYPES AND PROSTATE CANCER.

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INTRODUCTION AND OBJECTIVES: Prostatic calcification is associated with prostatic inflammation and there may be an association between inflammation and prostate cancer risk. We describe a novel calcification classification system on transrectal ultrasound (TRUS) and correlate this with histology and other parameters.

METHODS: A prospective, blinded study was designed of men undergoing TRUS and biopsy for standard indications using a B&K multifrequency probe with a standardised, reproducible technique. Representative sagittal and transverse images were recorded and analysed independently by a single experienced observer. The results were then compared with histology, IPSS, NIH prostatitis score, PSA and urinalysis. Statistical analyses were performed using Chi-square tests, tests for linear trend and non-parametric Kruskal - Wallis tests according to distribution.

RESULTS: A total of 483 men (aged 49-88) have been analysed thus far. There were 17 exclusions; 274 patients (58.8%) were diagnosed with prostate cancer, 88 patients (18.9%) with inflammation and 104 patients (22.3%) had benign pathology. Interface calcification was present in 42.3% of patients. Peripheral or transitional zone calcification was unusual (6.8% and 9.0% respectively). However 78.1% of the peripheral zone calcification group had cancer on histology ($p < 0.020$). There was no significant association with either NIH indices or IPSS categories or PSA.

CONCLUSIONS: Prevalence and characteristics of prostatic calcification have been described using this novel and practical classification. While interface calcification is common and not associated with any particular pathology, peripheral zone calcification appears to be strongly associated with prostate cancer.

Classification Zone	None	Interface	Transitional	Peripheral	p - value
Histology Group					
Benign	52-26.5%	45-22.8%	3-7.3%	4-12.5%	
Inflammation	31-15.8%	42-21.3%	12-29.3%	3-9.4%	
Cancer/Inflammation	113-57.7%	110-55.8%	26-63.4%	25-78.1%	$p < 0.020$

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2215 NOMOGRAM WITH "PSA ACCELERATION" PREDICTING HIGH GRADE PROSTATE CANCER

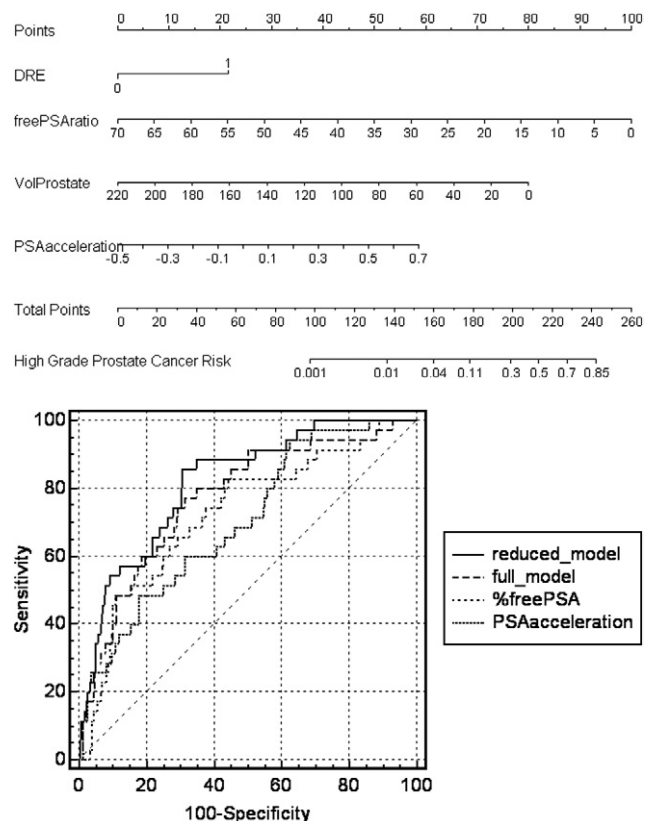
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INTRODUCTION AND OBJECTIVES: Many patients diagnosed with low grade prostate cancer may have indolent disease that may not benefit from immediate therapy. Our objective was to create a nomogram using PSA kinetics to predict high-grade prostate cancer (Gleason sum of 7 or more) (HG PCa).

METHODS: From a prospective database of twelve core prostate biopsies, we identified 630 men with at least 3 consecutive PSA measurements over a 2 year interval prior to biopsy. Least squares regression was used to calculate "PSA acceleration" (logPSA slope). Logistic regression was then used to predict HG PCa at biopsy using age, digital rectal examination findings (DRE), PSA, free to total PSA ratio (%fPSA), prostate volume and PSA acceleration. The population was randomly divided into two groups. A nomogram was developed in the training set and was then evaluated in the validation cohort.

RESULTS: Of the 630 men, 189 (30%) were diagnosed with prostate cancer on biopsy, and 75 (11.9%) had HG PCa. We applied backwards variable elimination to the full model, with the intent of identifying the most parsimonious and accurate model. In this model, all variables except age and PSA were significant predictors of HG PCa, and were included in the nomogram (Figure). In the validation population, the nomogram based on the parsimonious model had superior discrimination (AUC=0.817) compared to PSA, %fPSA, PSA density or PSA acceleration alone. Using a cutpoint of 17, the nomogram had a sensitivity of 85.7% and specificity of 69.3% for HGPCa.

CONCLUSIONS: The identification of clinically significant prostate cancer is essential to avoid overdiagnosis. We successfully developed a model to predict HG PCa including %fPSA, DRE findings, prostate volume and "PSA acceleration". Although the nomogram performed well in the internal validation, additional studies are warranted in external populations to confirm the clinical utility of this predictive tool.



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