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Original article

Racial variation in prostate needle biopsy templates directed anterior to the peripheral zone

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Abstract

Objectives: African Americans (AA) have been reported to have both increased incidence and increased aggressiveness of prostate cancer (PCa) located anterior to the peripheral zone (APZ). We sought to evaluate the utility of prostate biopsies directed toward the APZ in a predominantly AA cohort.

Methods and materials: We reviewed all patients with PCa found on biopsy schema that included needle biopsies directed at both the peripheral zone (PZ) and APZ from 2010 to 2014. Self-identified race was recorded for all patients. To evaluate the reliability of APZ-directed prostate biopsies, we performed pathologic secondary review of 25 radical prostatectomy specimens. A series of the Mann-Whitney *U* and Chi-square tests were used to compare variables.

Results: We identified 398 men, of which 277 (70%) were AA. Compared with non-AA, AA had more National Comprehensive Cancer Network-defined intermediate or high-risk (50% vs. 39%, $P = 0.25$) PCa. Most patients had PCa limited to the PZ only ($n = 190$) or in both the PZ and APZ ($n = 191$). For 17 patients (4%), PCa was limited only to the APZ core(s), 14 (5%) AA vs. 3 (2%) non-AA ($P = 0.24$). Most of these 17 patients ($n = 14$, 82%) had Gleason 6 disease.

Patients with PCa in both the PZ and APZ had higher serum prostate-specific antigen, prostate-specific antigen density, volume of disease, and increased grade and National Comprehensive Cancer Network category (all $P < 0.01$). Of these patients, there were no differences in race (AA = 135, 71% vs. non-AA = 56, 29%; $P = 0.48$). In only 21 men (11%), without racial variation, APZ tumor grade was greater than PZ. Radical prostatectomy and APZ-directed biopsies demonstrated a concordance rate of 80% (20/25), false positive rate of 8% (2/25), and false negative rate of 12% (3/25).

Conclusions: APZ-directed prostate biopsies are rarely the sole location of PCa and do not show a clear racial predilection. In those men with PCa identified in both regions, the APZ biopsy did not frequently change treatment recommendations. Biopsies directed at the APZ are not of greater benefit to AA than non-AA. Published by Elsevier Inc.

Keywords: Prostate cancer; African American; Prostate biopsy; Racial variation; Anterior to peripheral zone; Prostate biopsy

1. Introduction

More than a million prostate biopsies are performed yearly in the United States, resulting in greater than 230,000 new diagnoses of prostate cancer (PCa), making it the most common noncutaneous solid organ malignancy in men [1]. Most prostate biopsy techniques focus on the peripheral zone (PZ) of the prostate gland, where most tumors originate [2]. It is increasingly clear that a substantial number of tumors arise anterior to the peripheral zone (APZ) of the prostate [3]. This may be particularly important in Africans Americans (AA), who are both more likely to be diagnosed with, and more likely to die from, PCa than White Americans (non-AA). An emerging theory

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to explain this discrepancy is that AA harbor both an increased proportion of tumors and more aggressive tumors APZ of the prostate [4].

In 2014, the American Urologicals Association released specific recommendations on the optimal technique of prostate biopsy, stating that transition-zone biopsies do not substantially affect cancer detection rates, does not reduce negative predictive value, and does not improve predictive ability. However, they do note that comparing existing studies is challenging and that ethnicity or race may affect biopsy schema strategy. We hypothesized that for AA, who may harbor more APZ tumors, APZ-directed biopsies may be more likely to either be the sole location of a positive prostate biopsy or more likely to identify the highest-grade lesion. Ultimately a biopsy from this location may influence practitioner treatment recommendations. Thus, we sought to determine if prostate biopsies-directed APZ would be of particular value in diagnosing and characterizing PCa in AA. We aimed to test this hypothesis by reviewing the core location and Gleason findings in a racially diverse population of patients who were found to have PCa on biopsy that included cores directed at APZ.

2. Materials and methods

After obtaining Institutional Review Board approval, we retrospectively reviewed all patients without a previous diagnosis of PCa who underwent a transrectal ultrasound-guided (TRUS) biopsy at the Southeast Louisiana Veterans Health Care System (SLVHCS) between January 2010 and June 2014. In 2010, we altered our biopsy schema such that, in addition to a 12-core extended sextant PZ biopsy, we added 2 additional cores-directed APZ, making our template 14 cores (Fig.). Each core was individually placed in a separate specimen container for pathologic analysis. Cores were directed APZ by advancing the 18-gauge biopsy needle tip through the prostatic capsule approximately 0.5 cm in the mid-gland before firing. Fewer than 6% of patients had slight variations in this template in which 2 cores were taken from the APZ but only 10 cores were obtained from the PZ, and these patients were included in our analysis.

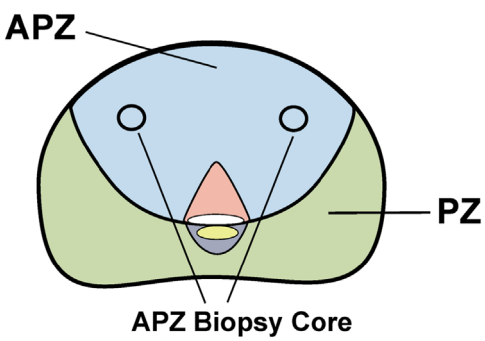

Fig. Biopsy template. In addition to a standard extended sextant prostate biopsy with 12 cores in the peripheral zone, 2 additional cores were aimed at tissue anterior to the peripheral zone (APZ).

Men were excluded if they had a previous diagnosis of PCa, had fewer than 12 cores taken, fewer than 2 cores from the APZ, underwent transperineal acquisition, or had a finding of high-grade prostate intraepithelial neoplasia or atypical small acinar proliferation suspicious for malignancy. All biopsies were performed at the SLVHCS in a racially diverse population. TRUS biopsies were performed for suspicion of PCa based on serum prostate-specific antigen (PSA) elevation or abnormality found on digital rectal examination. Over the course of the study the specimen handling and processing changed from 2 cores to a single container (i.e., right base lateral and medial) to a single core to a single container. Aggressiveness of disease was defined by Gleason grade, number of positive cores, highest percentage involvement of PCa in any core, clinical staging, and National Comprehensive Cancer Network (NCCN) classification. Self-identified race was recorded for all patients. All patients who did not identify themselves as AA were grouped together; greater than 95% of this group self-identified as white.

To confirm that APZ-targeted biopsies consistently and adequately sampled this region, we reviewed the radical prostatectomy (RP) specimens from a random sampling of 25 men who subsequently underwent this procedure at our institution. A secondary pathology review of the RP specimen was performed to determine the location of the tumor. The pathologists were blinded to the biopsy findings. A series of the Mann-Whitney *U*, Chi-square, and Fishers' Exact tests were used to compare variables.

3. Results

Between 2010 and 2014, 1,246 total biopsies (399 non-AA and 847 AA) were performed, in which there were 578 biopsies (398 AA and 180 non-AA) with PCa. Our total patient cohort consisted of 398 men found to have a TRUS prostate biopsy demonstrating PCa with at least 2 cores-directed APZ and a minimum of 10-cores directed at the PZ, and all but 1 patient (99.7%, $n = 397$) with 12 or more cores total. Most patients were AA ($n = 277$, 70%). When compared with non-AA, AA had similar median PSA values, but were younger, had smaller prostate volume, and had more aggressive PCa as defined by NCCN risk groups. A greater proportion of AA (vs. non-AA) had NCCN intermediate and high-risk (total 50% vs. 39%, $P = 0.31$) disease (Table 1). All but 11 men in this cohort had received no prior prostate biopsy, and none had a previous diagnosis of PCa.

Few patients (n = 17) had disease isolated in the APZ. Although a greater proportion were AA (5%) vs. non-AA (2%), small numbers prevented meaningful statistical comparisons. For those who did have disease isolated in the APZ, most patients had Gleason 3 + 3 disease (14/17), but