Baseline Basal Cell Hyperplasia Is not Associated With Baseline Lower Urinary Tract Symptoms, Baseline Clinical Prostatitis or Prostate Cancer in Repeat Biopsies

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OBJECTIVES To evaluate whether the presence of basal cell hyperplasia (BCH) in negative biopsies is associated with concurrent lower urinary tract symptoms (LUTS) and benign prostatic hyperplasia (BPH), clinical prostatitis, and future prostate cancer (PCa) in repeat prostate biopsy.

METHODS We performed a retrospective analysis of 6471 men, 50-75 years old with prostate-specific antigen between 2.5 and 10 ng/ml and prior negative biopsy who were enrolled in the Reduction by Dutasteride of PCa Events trial and underwent a 2-year repeat biopsy. The association between baseline BCH and risk of PCa, BPH/LUTS and clinical prostatitis measured at baseline were evaluated with logistic regression in uni/multivariable analysis, controlling for baseline patient characteristics.

RESULTS Among 6471 men enrolled, 84 (1.3%) had BCH in the baseline prostate biopsy. BCH was associated less chronic inflammation and more prostate atrophy ($P < 0.05$) and was unrelated to baseline patient characteristics. In both uni/multivariable analyses, BCH was not associated with PCa in repeat biopsy (univariable odds ratio [OR] = 0.98, 95% confidence interval [CI] = 0.53-1.82, $P > 0.05$; multivariable OR=1.15, 95% CI = 0.61-2.16, $P > 0.05$), BPH/LUTS (univariable OR = 1.13, 95% CI = 0.71-1.81, $P > 0.05$; multivariable OR = 1.20, 95% CI = 0.74-1.94, $P > 0.05$), or clinical prostatitis (univariable OR = 0.56, 95% CI = 0.18-1.81, $P > 0.05$; multivariable OR = 0.57, 95% CI = 0.18-1.83, $P > 0.05$).

CONCLUSION Among men undergoing repeat prostate biopsy with a baseline negative biopsy, BCH was associated with more histological atrophy and less chronic prostatitis, but was unrelated to LUTS/BPH, clinical prostatitis or future PCa risk. UROLOGY 129: 160–164, 2019. © 2019 Elsevier Inc.

Basal cell hyperplasia (BCH) is a benign histological finding characterized by the proliferation of prostatic acinar basal cells in the prostate.[1] It is a type of basal cell proliferation commonly associated with benign prostatic hyperplasia (BPH) and frequently found in the transitional zone of the prostate. Its prevalence ranges from 3.1%to 23% in needle biopsy specimens to 80% in whole prostates mounts.[1,2,3,4,5] Thorson et al demonstrated that BCH can also occur in the peripheral zone.[3] Proper identification of BCH is important given it may demonstrate architectural and cellular features resembling prostate cancer (PCa).[1] For example, previous studies have shown that a subtype of BCH called atypical or unusual BCH which presents with nuclear atypias, mitotic activity and abnormal architectural features can be difficult to differentiate from high-grade prostatic intraepithelial neoplasia (HGPIN) and PCa.[5]

Besides the potential to histologically mimic HGPIN and PCa, the clinical significance of BCH remains unclear with previous studies showing mixed results. For example, Henry *et al* found no correlation between BCH and age, PSA levels, prostate volume, medical treatments, or comorbidities among 141 subjects with BPH.[4] However, Anim et al found the prevalence of BCH to increase with age.[6] The same authors found BCH to be associated with prostate intraepithelial neoplasia. Conversely, Krystyna et al evaluating 537 prostate biopsy specimens found an

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inverse correlation between BCH and PCa.[7] Moreover, it remains unclear whether BCH in the peripheral zone of the prostate correlates with lower urinary tract symptoms (LUTS) or prostatitis symptoms. Therefore, we sought to evaluate whether the presence of BCH in baseline biopsies of men with an initial negative biopsy for PCa was associated with PCa at the 2-year repeat biopsy, baseline BPH/LUTS, and baseline clinical prostatitis among subjects in the REduction by DUtasteride of PCa Events (REDUCE) study. The REDUCE study was a multicenter controlled-placebo clinical trial analyzing the effects of dutasteride to reduce the incidence of PCa among subjects with negative baseline prostate biopsy.[8]

MATERIAL AND METHOD

Study Sample

We reviewed data from subjects enrolled in the REDUCE study where 50-75 years-old men with PSA levels between 2.5 and 10 ng/mL for ages 50-60 and between 3.5 and 10 ng/dL for ages 60-75 years, and only one negative prostate biopsy within 6 months prior enrollment were randomly assigned to receive either oral dutasteride 0.5 mg or placebo daily.[8] Subjects were followed every 6 months for 4 years. Men with International Prostate Symptom Scores (IPSS) ≥ 25, or ≥ 20 on alpha-blockers, prostate volume greater than 80 mL measured by ultrasonography, history of prostate surgery or previous detection of atypical small acinar proliferation, and HGPIN or PCa on prostate biopsy were not eligible. At least 10-core TRUS-guided prostate biopsy was performed in the second year as part of the study protocol. All baseline and 2-year repeat biopsies were read centrally (at Bostwick Laboratories). BCH consisted of 2 or more layers of proliferating basal cells at the periphery of the acini with or without prominent nucleolus. Architectural subtypes were not accessed. The presence of atrophy was defined as a single layer epithelium with cells with decreased cytoplasm and hyperchromatic nuclei, associated with small glandular acini and eventually stromal fibrosis. Chronic inflammation was defined as lymphocytic infiltrate with macrophages and plasma cell and acute prostatitis as neutrophilic infiltrate. Two independent pathologists evaluated all histological findings and discrepancies were settled by consensus. BCH, atrophy, and acute and chronic inflammation were coded as present or absent. Clinical prostatitis was defined as a positive response to CPSI (Chronic Prostatitis Symptom Index) question 1a (perineal pain) and/or question 2b (ejaculatory pain) and a total pain subscore of at least 4. BPH/LUTS was defined as an American Urological Association symptom score greater than 14 or medical treatment for BPH. Of the 8122 subjects enrolled in REDUCE, 6490 (80%) underwent a 2-year repeat prostate biopsy. Of these, we excluded 19 (<1%) due to missing data, resulting in a final study sample of 6471 men.

Statistical Analysis

The aims of the study were to evaluate the association between the presence of BCH in baseline biopsies with (1) the presence of PCa at 2-year repeat prostate biopsy, (2) baseline BPH/LUTS, and (3) baseline clinical prostatitis. Comparisons of baseline characteristics between the BCH groups (positive vs negative) were performed using chi-squared test for categorical variables and Student t test for continuous variables. Baseline characteristics evaluated were: age (continuous in years), PSA (continuous in ng/mL), body-mass-index (BMI, continuous in kg/m^2), PV (continuous in cm^3), digital rectal exam (DRE), race (White, Black, Asian, Hispanic, unknown), treatment arm (placebo or duasteride), family history of PCa (yes or no) and acute and chronic inflammations (yes or no), atrophy (yes or no). The association of BCH and PCa, clinical prostatitis and BPH/LUTS was evaluated using logistic regression in uni- and multivariable analyses controlling for baseline covariates (PSA, PV, age, DRE, race, BMI, and family history). Statistical analyses were performed using Stata 12.0 (StataCorp, College Station, TX). A $P < 0.05$ was considered to indicate statistically significant.

RESULTS

Baseline Patient Characteristics

Of the 6471 subjects included in the study, BCH was present in 84 (1.3%) cases. Subjects with BCH diagnosed in baseline biopsy were slightly younger and had less baseline chronic inflammation (odds ratio [OR] = 0.47, 95% confidence interval [CI] = 0.29-0.75, $P = 0.002$) but more acute inflammation (OR = 1.62, 95% CI = 1.00-2.63, $P = 0.050$) and more atrophy (OR = 3.78, 95% CI = 2.04-6.99, $P < 0.001$) than those without BCH. Others baseline characteristics were not significantly different between BCH groups (Table 1 and Supplementary Table 1).

BCH and BPH/LUTS

At baseline, the prevalence of BPH/LUTS was 27.3%. The prevalence of BPH/LUTS among those with and without BCH

Table 1. Association of baseline characteristics with basal cell hyperplasia

Variable	OR	95% CI	P
Age (years), median (SD)	0.97	0.94-1.00	0.072
PSA (ng/mL), median (SD)	0.98	0.88-1.08	0.608
BMI (Kg/m^2), median (SD)	1.03	0.99-1.08	0.162
Prostate volume (g), median (SD)	1.00	0.99-1.01	0.778
DRE, N (%)			
Normal	*ref.*	–	–
Abnormal	1.47	0.61-3.86	0.361
Treatment arm, N (%)			
Placebo	*ref.*	–	–
Dutasteride	1.41	0.95-2.10	0.091
FH prostate cancer			
No	*ref.*	–	–
Yes	0.90	0.48-1.66	0.728
Region, N (%)			
North America	*ref.*	–	–
Europe	1.34	0.81-2.20	0.255
Other	1.25	0.63-2.46	0.521
Acute inflammation, N (%)			
Absent	*ref.*	–	–
Present	1.62	1.00-2.63	0.050
Chronic inflammation, N (%)			
Absent	*ref.*	–	–
Present	0.47	0.29-0.75	0.002
Atrophy, N (%)			
Absent	*ref.*	–	–
Present	3.78	2.04-6.99	<0.001

BMI: body-mass index; CI: confidence interval; FH: family history; OR: odds ratio; PSA: prostate-specific antigen; *ref.*: reference.

Table 2. Association of basal cell hyperplasia and benign prostate hyperplasia

Basal Cell Hyperplasia	BPH		Univariable			Multivariable		
	Absent	Present	OR	95% CI	*P*	OR	95%CI	*P*
Absent	4,648 (72.8)	1,739 (27.2)	*ref.*	–	–	*ref.*	–	–
Present	59 (70.2)	25 (29.8)	1.13	0.71-1.81	0.604	1.20	0.74-1.94	0.459

BPH: benign prostate hypertrophy; CI: confidence interval; OR: odds ratio; *ref.*: reference.

Table 3. Association of basal cell hyperplasia and clinical prostatitis

Basal Cell Hyperplasia	Clinical Prostatitis		Univariable			Multivariable		
	Absent	Present	OR	95% CI	*P*	OR	95% CI	*P*
Absent	3,988 (91.5)	372 (8.5)	*ref.*	–	–	*ref.*	–	–
Present	57 (95.0)	3 (5.0)	0.56	0.18-1.81	0.336	0.57	0.18-1.83	0.343

CI: confidence interval; OR: odds ratio; *ref.*: reference.

was 29.8% and 27.2%, respectively. In either uni- and multivariable analyses, baseline BCH was not associated with baseline BPH/LUTS (univariable OR = 1.13, 95% CI = 0.71-1.81, *P* = 0.604 multivariable OR = 1.20, 95% CI = 0.74-1.94, *P* = 0.459; Table 2).

BCH and Clinical Prostatitis

The overall prevalence of clinical prostatitis at baseline was 8.5%. The prevalence of clinical prostatitis among those with and without BCH was 5.0% and 8.5%, respectively. In either uni- and multivariable analyses, baseline BCH was not associated with baseline clinical prostatitis (univariable OR = 0.56, 95% CI = 0.18-1.81, *P* = 0.336; multivariable OR = 0.57, 95% CI = 0.18-1.83, *P* = 0.343; Table 3).

BCH and PCa

The overall incidence of PCa at 2-year prostate biopsy was 14.7%. The incidence of PCa in subjects with or without BCH was 14.5% and 14.7%, respectively. In either uni- or multivariable analyses, baseline BCH was not associated with PCa at the 2-year biopsy (univariable OR = 0.98, 95% CI = 0.53-1.82, *P* = 0.952; multivariable OR = 1.15, 95% CI = 0.61-2.16, *P* = 0.665; Table 4).

DISCUSSION

Basal cell hyperplasia is the most common type of prostate basal cell proliferation, a histological entity that can range from a benign condition to basal cell carcinoma.[9] Although BCH is usually benign and more frequently found in the transitional zone associated with BPH, it can also be found in the peripheral zone of the prostate, where PCa is more prevalent.[3,10] Given the cytological and architectural changes seen in some BCH cases, it can occasionally be mistaken for HGPIN or PCa.[11] Previous studies evaluating the association of BCH with HGPIN and PCa have shown diverting results. Moreover, the clinical relevance of BCH on prostate biopsy specimens as it relates to LUTS, prostatitis and even PCa has not been studied in earnest. Therefore, we analyzed whether BCH on baseline prostate biopsies was associated with PCa at 2-year repeat prostate biopsy in the REDUCE study.[8] We also studied whether baseline BCH was associated with baseline BPH/LUTS and clinical prostatitis. We found baseline BCH was not associated PCa risk at 2-year biopsy, clinical BPH/LUTS or clinical prostatitis. However, baseline BCH was associated with more baseline prostate atrophy and less chronic inflammation.

In our study, the prevalence of BCH was quite low (1.3% of baseline prostate biopsies that were negative for PCa). This finding contrasts with previous studies reporting a higher BCH prevalence, ranging from 3.1% to 23%.[1,2,5] This difference likely stem from the fact that most of previous studies analyzed samples from transitional zone, where BPH is commonly seen; while we evaluated almost exclusively the peripheral zone of the prostate. Conversely, a previous study of biopsy cores from the peripheral zone found an 11% prevalence of BCH. In that study, the authors commented that some cores might have been taken from transitional zone due to a sizable number of biopsies performed in large volume prostates.[5] Contrariwise, in our study we excluded men with larger prostates (prostate volume >80 cm^3) and severe LUTS, which may explain the lower prevalence of BCH due to the absence of very large prostates. Thus, it seems the prevalence of BCH in prostate biopsies varies according to the sampled area and gland characteristics such as volume and presence of BPH.

Table 4. Association of basal cell hyperplasia and prostate cancer

Basal Cell Hyperplasia	Prostate Cancer		Univariable			Multivariable		
	Absent	Present	OR	95% CI	*P*	OR	95% CI	*P*
Absent	5306 (85.3)	914 (14.7)	*ref.*	–	–	*ref.*	–	–
Present	71 (85.5)	12 (14.5)	0.98	0.53-1.82	0.952	1.15	0.61-2.16	0.665

CI: confidence interval; OR: odds ratio; *ref.*: reference.

The factors associated with BCH are not entirely understood. For example, Henry et al found no association between BCH and age while Anim et al found the prevalence of BCH to increase with aging.[4,6] Conversely, Thorson et al found an association between BCH and younger age which agrees with our findings.[3] Other patient factors such as race, BMI, PSA levels, DRE, prostate volume, and geographic region were not associated with BCH. Similar results were obtained by Henry et al who found no correlation between BCH and PSA levels, prostate volume, medical treatments, or comorbidities.[4] However, in our study BCH was associated with more histological prostate atrophy and acute inflammation and less chronic inflammation. Contrariwise, Thorson et al, found BCH to be associated with lymphocytic (chronic) inflammatory infiltrate in most cases.[3] Given the conflicting results on the factors associated with BCH, further studies evaluating the potential causes of BCH are warranted.

The relationship of BCH in prostate biopsies with LUTS and prostatitis-related symptoms is unknown. Yang et al postulated that basal cell proliferation was present in almost all subjects who underwent surgery for BPH/LUTS while Henry et al found BCH in only 13 subjects of the 141 prostate TURP and open prostatectomy specimens evaluated.[4,12] In our study, we found no association between BCH in the peripheral zone with prostate volume or IPSS score. This suggests that BCH found in the peripheral zone is not associated with BPH or LUTS while BCH in the transitional zone may be associated with BPH and LUTS, though this requires further study.

Previous studies have suggested BCH could be a metaplastic reaction to injury and in some cases may progress to basal cell carcinoma.[13,14] However, this hypothesis has been contested in multiple studies. For example, Thorson et al demonstrated that the presence of BCH in peripheral zone was associated with lower PCa risk.[3] In addition, they did not find any topographical association between BCH and PCa in prostate specimens. Similarly, a study by Benett et al failed to demonstrate PCa development in patients diagnosed with BCH.[14] In our study, PCa at the 2-year repeat biopsy was unrelated to baseline BCH. Thus, although it seems that BCH is unrelated to PCa, further studies evaluating the molecular changes associated with BCH and how they correlated with prostate carcinogenesis are needed.

Our study has several strengths including its large, multicentric sample with central pathology review by 2 independent pathologists. Nonetheless, it has some limitations. First, subjects with severe LUTS (IPPS >= 25 or 20 among those on alpha-blocker) and prostate volume >80 cm^3 were excluded and only men with baseline PSA values between 2.5 and 10 ng/dl were included. Second, those with atypical small acinar proliferation, HGPIN, or previous prostate surgery were also excluded. Third, we did not evaluate subtypes of BCH, including typical and atypical. Finally, the prevalence of BCH was low, which reduced our statistical power.

CONCLUSION

Among men undergoing repeat prostate biopsy, BCH was associated with more histological atrophy and less chronic prostatitis. BCH was not associated with LUTS/BPH, clinical prostatitis or PCa. Thus, given the limited clinical relevance of BCH, it may be excluded from routine prostate biopsy pathology reports.

SUPPLEMENTARY MATERIALS

Supplementary material associated with this article can be found in the online version at https://doi.org/10.1016/j.urology.2019.02.034.

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EDITORIAL COMMENT

Repeat biopsies in prostate disease are notoriously difficult to acquire, but provide a wealth of data linking phenotype with disease progression.

The REDUCE trial is a rare longitudinal clinical trial that performed prostate biopsy at baseline and again at 2 and 4 years to determine the effect of dutasteride on prostate cancer

prevention.[1] The authors performed a retrospective analysis of peripheral zone needle biopsies from 6471 men enrolled in the REDUCE trial to determine the association between baseline basal cell hyperplasia (BCH) and the risk of (1) prostate cancer, (2) benign prostatic hyperplasia with lower urinary tract symptoms, or (3) prostatitis. In short, BCH was not predictive of any clinically meaningful disease progression at 2 years, prompting the suggestion that this rare phenotype should not even be noted by pathologists.

It is important to document these negative results, but with several caveats noted. The incidence of BCH in needle biopsies from this REDUCE cohort was extremely low (1.3%), with other cohorts demonstrating an incidence of 3.1% in needle biopsies to 23% in whole mounts.[2] Our own study of BCH in transition zone tissue from patients with BPH/LUTS undergoing TURP or simple prostatectomy tissue showed an incidence of 8%, but the total volume of BCH foci was highly variable between patients.[3] The low incidence and sporadic appearance of BCH foci in the peripheral zone may make its random detection in biopsies an underestimate, and the relatively short 2-year follow-up may be insufficient to detect clinically meaningful differences in relation to prostate cancer progression. On this point, it is unclear why the biopsies from the 4-year follow-up in the REDUCE trial were not used instead. It is also difficult to extrapolate the relationship between BCH detected by biopsy in the peripheral zone and symptoms due to transition zone BPH. However, the conclusion that BCH is not related to age, prostate volume, LUTS, or prostatitis does largely agree with our own study performed on larger specimens from the transition zone.[3]

Based on a transcriptional profile of basal cells from patients with BCH compared to basal cells from patients without BCH, we established that BCH is likely a metaplastic event.[3] This study suggests that the BCH phenotype is clinically meaningless and should be ignored. Perhaps future studies with a higher incidence and longer follow-up will alter this opinion, but it an important observation nonetheless.

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AUTHOR REPLY

Several nonmalignant histological prostate findings have been shown to be of clinical relevance. For example, we and others have shown chronic histological prostate inflammation in needle biopsies is associated with worsening prostatitis- and benign prostate hyperplasia (BPH)-related symptoms over time.[1,2] While basal cell hyperplasia (BCH) in the transitional zone has been associated with BPH in prior studies, the clinical significance of BCH in needle biopsies of the peripheral zone has not been previously studied in earnest.[3,4] Although limited by a low prevalence of BCH (1.3%), the current study is one of the largest analysis of BCH and it was unable to identify any association between BCH and prostate cancer, BPH or prostatitis-related symptoms. The lower BCH prevalence in our study likely stem from the fact that: (1) our biopsies targeted the peripheral zone only, and (2) subjects with very large prostates and/or severe urinary symptoms were excluded. Moreover, data from the 4-year repeat prostate biopsies were indeed analyzed but not included in the final manuscript given the results were similar to the ones obtained using 2-year biopsy data. While it is conceivable that future studies with larger BCH samples may identify correlations between BCH and clinically meaningful outcomes, the current relevance of BCH seems to rely solely on being a differential diagnosis for high-grade prostatic intraepithelial neoplasia and prostate adenocarcinoma and, as such, may be omitted from routine pathological prostate needle biopsy reports.

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