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DIABETES TREATMENT AND PROGRESSION OF BENIGN PROSTATIC HYPERPLASIA IN COMMUNITY DWELLING BLACK AND WHITE MEN

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Abstract

Objectives—Diabetes has been associated with benign prostatic hyperplasia (BPH) and lower urinary tract symptoms (LUTS) in aging men. We conducted a study to determine if diabetes treatment was associated with BPH/LUTS and progression in black and white men.

Methods—Using the *Olmsted County Study of Urinary Symptoms and Health Status among Men* (OCS) and the *Flint Men's Health Study* (FMHS), we examined how use of medical therapy (e.g., insulin regimens, oral hypoglycemics, etc.) related to changes in LUTS severity, maximum urinary flow rate measured by uroflowmetry, prostate volume determined by transrectal ultrasound, and serum PSA concentrations.

Results—Of the 2,226 men participating in the OCS and the FMHS, 186 men reported a history of diabetes, 76.9% of which were treated with medical therapy. Overall, men with diabetes had significantly greater odds of moderate/severe LUTS (age- and race-adjusted OR=1.37, 95% CI=1.00, 1.87) compared to non-diabetics. However, among diabetic men, those not taking medications had higher odds of moderate/LUTS than those taking medications. This association among men not taking medications was seen for five of the seven individual symptoms. Prostate volume and PSA were not significantly associated with diabetes treatment. No significant differences were observed for annual change in BPH characteristics by diabetes treatment status.

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Conclusions—These findings suggest that the presence of diabetes and subsequent poor glycemic control may be less related to prostate growth and more to the dynamic components of lower urinary tract function. Further evaluations of the associations between glycemic control and BPH progression are warranted.

Keywords

diabetes; BPH; LUTS; diabetes; men; aging

INTRODUCTION

Benign prostatic hyperplasia (BPH) is the most common benign neoplasm in American men. Marked by the progressive development of lower urinary tract symptoms (LUTS), its prevalence increases with age and over 6.5 million Americans meet criteria for treatment. In 2010 alone, 4.5 million visits to physicians' offices will be made for BPH and over 87,000 BPH-related surgeries will be performed. Exclusive of medical therapy, direct costs of BPH treatment exceed $1.3 billion annually.[1]

To help lessen the disease's substantial public health burden, there is a need for interventions that prevent the development of BPH, as well as symptom progression in men with mild/moderate LUTS. Accumulating evidence indicates an association between diabetes and BPH. In a previous cross-sectional baseline comparison[2], we observed that diabetic men reported more moderate/severe American Urological Association Symptom Index (AUASI) scores than non-diabetic men. Insofar as glycemic control is associated with a decreased risk of BPH, high-risk groups could be targeted with diabetes treatment plans to reduce the incidence of this common condition.

To explore this possibility, we used data from two cohort studies of community-dwelling men: the *Olmsted County Study of Urinary Symptoms and Health Status among Men* (OCS) and the *Flint Men's Health Study* (FMHS). We leveraged the longitudinal nature of the OCS and the FMHS to determine whether treatment for diabetes (glycemic control) was associated with the progression of BPH in two racially diverse populations.

MATERIAL AND METHODS

Study Population

Details on subject selection for both the OCS and FMHS have been previously published.[3,4] Briefly, the OCS and FMHS are population-based, prospective cohort studies established to evaluate the natural history of BPH in white and black male residents of Olmsted County, Minnesota and Genesee County, Michigan, respectively.

In the OCS, 2,115 of 3,874 eligible white men aged 40-79 years in 1990 without history of prostate cancer or surgery or other conditions known to interfere with voiding including diabetic neuropathy leading to lower limb amputation, completed the self-administered AUASI.[5] A detailed urologic examination that included uroflowmetry, transrectal ultrasound (TRUS), and serum prostate-specific antigen (PSA) measurement was conducted on a 25% random subsample (476 out of 537, 89%). All of the men in the cohort have been followed biennially since 1990. At each round of follow-up, all men completed the same protocol. Men who died or were lost to follow-up during the course of the study were replaced during rounds 2 and 3, resulting in a total of 2,447 study participants and 634 subsample participants to date.

Using the same criteria and protocol described above, 730 of 943 eligible black men completed an interview-administered questionnaire in 1996 in the FMHS. Of these, 369 men underwent the comprehensive urologic examination, which included, as in the OCS, uroflowmetry, TRUS, a serum PSA measurement and self-administered AUASI and were deemed to be free of prostate cancer. Four years after baseline (2000), the 369 men who participated in the baseline clinical exam were re-contacted and invited to complete the same study protocol described above. Of the 369 men, 186 (50%) were available and agreed to participate at follow-up.

The study population for the current analyses is limited to data from Rounds 4 (1996) and 6 (2000) of the OCS and baseline (1996) and follow-up (2000) of the FMHS to provide temporal comparability. 2,140 men participated in Round 4 of the OCS. Those with prostate cancer and/or treatment prior to Round 4 were removed for a total of 1,863 white men for the current report. Similarly, of the 369 black men who participated at baseline in the FMHS, 363 were free of prostate cancer and/or treatment prior to their baseline visit and included in the current report for a total sample of 2,226 men (1,863 white, 363 black).

Measurements

Diabetes—Information on diabetes was gathered by questionnaire in both the OCS and FMHS at baseline. Participants were asked whether they had ever been diagnosed by a physician to have diabetes mellitus and in which year they were diagnosed. Treatment for diabetes was defined by patient report of use of oral diabetes medications or insulin.

Benign Prostatic Hyperplasia—The primary endpoints included the following clinical markers of BPH: LUTS severity, maximum urinary flow rate, prostate volume, and serum PSA concentrations. Specifically, LUTS severity was measured by the AUASI score via self-administered questionnaires in both the OCS and the FMHS. Prostate volume (ml) determined by transrectal ultrasound, maximum urinary flow rate (ml/sec) measured by uroflowmetry and serum PSA (ng/ml) concentrations were collected during the clinical exam portions of the two studies. Although no single surrogate measure provides a definitive non-histologic diagnosis of BPH, previous studies have demonstrated that these measures have adequate construct and predictive validity for BPH.[6]

Statistical Analysis

Since the FMHS urologic measurements were collected in 1996 and 2000, the corresponding 1996 and 2000 OCS measurements were used in these analyses as the baseline and four year follow-up measures. Characteristics of the study populations at baseline were compared by diabetes status using chi-square tests. Odds ratios and corresponding 95% confidence intervals were calculated to examine the associations of diabetes treatment status (non-diabetics, diabetics taking medications, and diabetics not taking medications) with baseline BPH/LUTS characteristics. Multivariable logistic regression models were used to adjust for age and race. The empirical distribution of annual change (points/year) in AUASI score was calculated by dividing the difference between the baseline and four-year follow-up AUASI score by the number of years between the measurements. Annual percent change for maximum urinary flow rate, prostate volume, and PSA concentration was calculated by dividing the difference between baseline and four-year follow-up measure by the product of the baseline measure and the time between the two measures multiplied by 100 (for percent). Tests for differences were examined across diabetes treatment categories.

RESULTS

Among the 2,226 total participants (1,863 white and 363 black men), 186 (8.4%) had a self-reported history of diabetes (Table 1). Mean age at baseline was 62.5 years (standard deviation [SD], 10.4) and 57.5 years (SD, 10.1) in those with and without diabetes, respectively ($p<0.001$). Overall, 78.8% of men were overweight/obese (Body Mass Index (BMI) ≥25 kg/m^2), and men with diabetes were more likely to be overweight compared to men without diabetes (Table 1). Black men were also more likely to have a history of diabetes than white men ($p<0.001$) Data on the incidence of LUTS severity and progression overall by age and race can be found in the Appendix.

In bivariate analyses, we compared three groups of men: men taking medications to treat diabetes, men not taking medications to treat their diabetes, and non-diabetics. Overall, men with diabetes had significantly greater odds (age- and race-adjusted odds ratio (OR) = 1.37, 95% confidence interval (CI) = 1.00, 1.87) of moderate/severe LUTS (AUASI score >7) compared to non-diabetics. However, diabetic men who were not taking medications had higher odds of moderate/severe LUTS than diabetic men who were taking medications (Table 2). Specifically, the frequency of irritative symptoms (AUASI score >3 for symptoms of urgency, frequency and nocturia) was significantly higher among diabetic men with the greatest impact observed among diabetic men not taking medications (age- and race-adjusted OR = 2.04, 95% CI = 1.08, 3.86) compared to non-diabetics. In multivariable analyses adjusted for age and race, prostate volume and total PSA were not significantly associated with diabetes treatment status at baseline (Table 2). When assessing associations between diabetes and individual symptoms (Table 3), five of the seven individual symptoms were significantly (or marginally significant) higher among diabetic men not taking medications (age- and race-adjusted OR range from 1.82 to 2.40). Only the symptom of nocturia was also significantly higher among diabetics taking medications (age- and race-adjusted OR = 2.22, 95% CI = 1.52, 3.23) compared to non-diabetics. Finally, no significant differences were observed for annual change in BPH characteristics by diabetes treatment status (Table 4).

COMMENT

Type 2 diabetes, which affects 90%–95% of people with diabetes, has been associated with bladder dysfunction, typically resulting in impairment of the detrusor.[7,8] Impaired detrusor function results in a lower maximum flow rate for any given level of bladder outlet resistance and can increase post-void residual and LUTS severity.[7] BPH is also characterized by its presentation of LUTS, including a reduced maximum urinary flow rate and increased post-void residual. The underlying pathophysiology, however, is different since BPH does not primarily impair detrusor function but enhances bladder outlet resistance via static and dynamic components.[7] In a baseline examination of the current combined cohort, we previously reported significant differences in the presence of irritative LUTS in men with diabetes compared to men without diabetes. The current report expands on this analysis to assess the potential for the effect of glycemic control on BPH/LUTS measures, by examining associations between treatment for diabetes and measures of BPH and LUTS progression in community-dwelling black and white men. While several studies have examined the association between diabetes and BPH, findings have been inconsistent. A series of cross-sectional studies from Sweden observed that physician-diagnosed Type 2 diabetes, treated hypertension, obesity, low high-density lipoprotein-cholesterol levels, and high insulin levels were significantly associated with the presence of BPH in a consecutive series of patients with LUTS referred for surgery.[9–11] Furthermore, the Massachusetts Male Aging Study[12], the FMHS[13], and others[14,15] have consistently reported diabetes or glucose levels to be significantly associated with an increased risk of LUTS.

The positive associations described between measures of diabetes and BPH, however, have not been consistently observed across studies. Specifically, Boon, et al. examined individuals with physician-diagnosed diabetes and LUTS compared to individuals with LUTS only and found little difference in prostate volume, maximum urinary flow rate, and post-void residual volume.[16] This study, however, relied on a control group from a referral population that did not meet the specified exclusion criteria for BPH, and thus likely underestimated the effect of diabetes on LUTS. Furthermore, in contrast to their finding for BPH surgery, the Normative Aging Study found a non-significant inverse association between diabetes and clinical BPH.[17] Finally, in several reports using data from the OCS, Burke, et al.[18] and the baseline comparison of the current combined cohort, Sarma, et al.[19] observed that diabetic men reported more moderate/severe AUASI scores than did non-diabetic men. However, we found no differences in prostate volume suggesting, perhaps, that the presence of diabetes may be less directly associated with prostate growth and more closely associated with the dynamic components of lower urinary tract function.

Importantly some of the aforementioned studies utilized markers of BPH (e.g., transurethral resection of the prostate) to define disease. These markers can be a poor endpoint for LUTS in diabetic men whose LUTS could be a result of bladder dysfunction.[20] Furthermore, the failure to differentiate LUTS from BPH, along with the lack of inclusion of additional clinical markers more specific to BPH may have contributed to the confusing evidence now seen in the literature.[20] Finally, these studies were limited by their inclusion of primarily white men, lack of population-based samples,[21,22] and cross-sectional designs which the current examination of the FMHS and OCS overcome.

In this study, we observed that diabetes was significantly associated with increased symptom severity and that this effect was most prominent among men who reported not taking medications for their diabetes. Increased prostate volume, increased serum PSA levels and decreased urinary flow rates were not significantly associated with diabetes treatment status and suggest that there is no strong evidence for an association between diabetes treatment and BPH across measures. Given the lack of evidence with measures more specific to prostate disease, the association observed between diabetes and LUTS is likely attributed to diabetic neuropathy and is largely driven in this study by the significant association observed specifically between diabetes and nocturia. Although nocturia may be a consequence of changes in bladder reservoir function and/or kidney function secondary to urinary tract obstruction, nocturia has been associated with diabetes in numerous reports.[23–25]

There are several mechanisms by which diabetes may influence BPH. The first is via changes in insulin concentrations which may, in turn, influence sex hormone concentrations,[26] sympathetic nerve activity, and/or the insulin-like growth factor axis and affect the growth of the prostate.[10,27] In addition, poorly controlled diabetes can cause osmotic diuresis which may be associated with urinary frequency and nocturia and also affect LUTS via neuropathic mechanisms, influencing both motor and sensory nerves.[28] This is supported by our findings that diabetic men who were not taking medications had higher odds of moderate/severe LUTS compared to men without diabetes, including five of the individual symptoms. These associations were not seen in diabetic men who were taking medications with the exception of the symptom of nocturia.

In addition, we did not observe statistically significant associations between diabetes treatment and more specific measures of BPH. However, the magnitude and direction of the associations observed across the spectrum of BPH measures suggest that potentially, diabetes may influence not only the dynamic components of lower urinary tract function via the bladder but may even influence prostatic growth. This is evidenced specifically in the

marginal positive association observed between diabetes and prostate volume, particularly among diabetic men not on medications. This observation could be explained, in part, by the relationship between increasing insulin concentrations and insulin-like growth factor (IGF) bioavailability which has be found to be associated with prostate growth.[29,30] Furthermore, it is possible that there are conflicting impacts of glycemic control on the prostate and bladder that would result in inconsistent findings across measures of BPH. If diabetes slows down prostate growth via testosterone and growth factors, it might reduce the risk of obstructive LUTS but not necessarily mask the beneficial effects of glycemic control on the bladder, which would present via irritative symptoms.

As both BPH and diabetes are highly prevalent conditions of significant burden in the US, the potential of prostate and bladder disease as complications of poorly controlled diabetes warrants further investigation in study populations with larger samples of men with diabetes and identifies a target for primary and secondary prevention.

Although this is one of the first studies to examine the association between diabetes treatment and progression of clinical markers of BPH in a multi-ethnic population-based sample of men, there are several limitations that should be considered. First, this study relies on self-reported history of physician-diagnosed diabetes and its treatment, which may result in the inclusion of individuals with diabetes in the control group or vice versa. However, this misclassification is not likely to be differential by markers of BPH and would most likely result in an underestimation of the association between BPH markers and diabetes treatment. Second, although the findings reveal positive associations between diabetes treatment and various clinical markers of BPH, we cannot exclude the possibility of chance as an explanation for our findings as the confidence intervals for the multivariable estimates include one. This could likely be attributed to the limited sample size available among the clinical subset over time. However, this is one of few studies with comprehensive clinical data regarding measures of BPH and estimating the magnitude of the association between diabetes treatment, and BPH progression is an important first step in determining whether relationships indeed exist. These potential limitations are offset by the strengths of this study, including a longitudinal, population-based multi-ethnic sample of men with comprehensive set of clinical markers of BPH.

CONCLUSION

In this community-based study of BPH and diabetes, we have demonstrated associations between diabetes treatment and increased LUTS, particularly irritative LUTS severity. Moreover, the magnitude of the association between irritative LUTS and diabetes was most pronounced in diabetic men who were not taking medications. Furthermore, there was no strong evidence for an association between diabetes and BPH across measures more specific to BPH (i.e. prostate volume, PSA level). Taken together, our findings suggest that the presence of diabetes and subsequent poor glycemic control may be less related to prostate growth and more related to the dynamic components of lower urinary tract function. Further evaluations of the associations between diabetes, glycemic control, and BPH progression are warranted.

Acknowledgments

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Table 1

Overall distribution of characteristics in men with and without diabetes at baseline

Characteristic	No Diabetes (N=2,040) N (%)*	Diabetes (N=186) N (%)*	p-value
Age (years)			<0.001
40–49	610 (29.90)	23 (12.37)	
50–59	696 (34.12)	57 (30.65)	
60–69	438 (21.47)	52 (27.96)	
70+	296 (14.51)	54 (29.03)	
Body mass index (kg/m^2)			<0.001
Normal (<25)	409 (22.00)	22 (12.79)	
Overweight (25–29)	892 (47.98)	62 (36.05)	
Obese (≥30)	558 (30.02)	88 (51.16)	
Race			<0.001
White (OCS cohort)	1,741 (85.34)	122 (65.59)	
Black (FMHS cohort)	299 (14.66)	64 (34.41)	
Diabetes Medication Use			
Yes			
White (OCS Cohort)	-	97 (67.83)	
Black (FMHS Cohort)	-	46 (32.17)	

* Percents based on non-missing values

Table 2

Association between diabetes medication use and BPH/LUTS characteristics in community-dwelling white and black men at baseline

Characteristic	No Diabetes (N=2,040) N (%)*	Diabetes (no meds) (N=43) N (%)*	Diabetes (meds) (N=143) N (%)*
LUTS severity			
Mild/none (AUASI score≤7)	1,276 (62.92)	19 (45.24)	73 (51.77)
Moderate/severe (AUASI score>7)	752 (37.08)	23 (54.76)	68 (48.23)
Unadjusted OR (95% CI)	reference	2.05 (1.11, 3.80)	1.58 (1.12, 2.23)
Age- and race-adjusted OR (95%CI)	reference	1.77 (0.94, 3.31)	1.26 (0.89, 1.80)
Obstructive symptom score			
≤4	1,396 (68.97)	23 (54.76)	96 (68.09)
>4	628 (31.03)	19 (45.24)	45 (31.91)
Unadjusted OR (95% CI)	reference	1.84 (0.99, 3.40)	1.04 (0.72, 1.50)
Age- and race-adjusted OR (95% CI)	reference	1.73 (0.92, 3.23)	0.89 (0.61, 1.30)
Irritative symptom score			
≤3	1,217 (60.04)	16 (37.21)	63 (44.37)
>3	810 (39.96)	27 (62.79)	79 (55.63)
Unadjusted OR (95% CI)	reference	2.54 (1.36, 4.74)	1.88 (1.34, 2.65)
Age- and race-adjusted OR (95% CI)	reference	2.04 (1.08, 3.86)	1.46 (1.02, 2.08)
Maximum urinary flow rate (ml/sec)†			
≤12	427 (84.55)	14 (82.35)	27 (69.23)
<12	78 (15.45)	3 (17.65)	12 (30.77)
Unadjusted OR (95% CI)	reference	1.17 (0.33, 4.18)	2.43 (1.18, 5.01)
Age- and race-adjusted OR (95% CI)	reference	0.96 (0.26, 3.51)	1.99 (0.91, 4.34)
Prostate volume (ml)†			
≤30	406 (64.75)	12 (48.00)	30 (50.85)
>30	221 (35.25)	13 (52.00)	29 (49.15)
Unadjusted OR (95% CI)	reference	1.99 (0.89, 4.44)	1.78 (1.04, 3.04)
Age- and race-adjusted OR (95% CI)	reference	1.85 (0.80, 4.27)	1.53 (0.86, 2.72)
Total PSA (ng/ml)†			
≤2.5	595 (86.61)	21 (77.78)	51 (80.95)
>2.5	92 (13.39)	6 (22.22)	12 (19.05)
Unadjusted OR (95% CI)	reference	1.85 (0.73, 4.70)	1.52 (0.78, 2.96)
Age- and race-adjusted OR (95% CI)	reference	1.47 (0.54, 3.97)	0.93 (0.45, 1.92)

*Percents based on non-missing values

†Measures on men with in-clinic examinations only (414 OCS men, all 363 FMHS men: 687 No Diabetes, 27 for Diabetes (no meds), and 63 for Diabetes (meds))

Table 3

Association between diabetes medication use and individual symptoms in community-dwelling white and black men at baseline

Characteristic	No Diabetes (N=2,040) N (%)*	Diabetes (no meds) (N=43) N (%)*	Diabetes (meds) (N=143) N (%)*
Frequency symptom score			
≤1	1,337 (66.19)	20 (46.51)	88 (61.97)
>1	683 (33.81)	23 (53.49)	54 (38.03)
Unadjusted OR (95% CI)	reference	2.25 (1.23, 4.13)	1.20 (0.85, 1.71)
Age- and race-adjusted OR (95% CI)	reference	1.90 (1.03, 3.51)	1.02 (0.72, 1.47)
Urgency symptom score			
≤1	1,369 (67.67)	23 (54.76)	90 (63.83)
>1	654 (32.33)	19 (45.24)	51 (36.17)
Unadjusted OR (95% CI)	reference	1.73 (0.94, 3.20)	1.19 (0.83, 1.69)
Age- and race-adjusted OR (95% CI)	reference	1.82 (0.97, 3.42)	1.08 (0.75, 1.57)
Nocturia symptom score			
≤1	1,532 (76.03)	20 (46.51)	67 (48.20)
>1	483 (23.97)	23 (53.49)	72 (51.80)
Unadjusted OR (95% CI)	reference	3.65 (1.99, 6.70)	3.41 (2.41, 4.83)
Age- and race-adjusted OR (95% CI)	reference	2.40 (1.26, 4.60)	2.22 (1.52, 3.23)
Incomplete emptying symptom score			
≤1	1,604 (79.25)	26 (61.90)	106 (75.18)
>1	420 (20.75)	16 (38.10)	35 (24.82)
Unadjusted OR (95% CI)	reference	2.35 (1.25, 4.42)	1.26 (0.85, 1.88)
Age- and race-adjusted OR (95%CI)	reference	2.01 (1.06, 3.81)	1.05 (0.70, 1.58)
Intermittency symptom score			
≤1	1,515 (74.74)	23 (54.76)	110 (78.01)
>1	512 (25.26)	19 (45.24)	31 (21.99)
Unadjusted OR (95% CI)	reference	2.45 (1.32, 4.53)	0.83 (0.55, 1.26)
Age- and race-adjusted OR (95% CI)	reference	2.33 (1.25, 4.37)	0.73 (0.48, 1.11)
Straining symptom score			
≤1	1,768 (87.61)	35 (85.37)	121 (86.43)
>1	250 (12.39)	6 (14.63)	19 (13.57)
Unadjusted OR (95% CI)	reference	1.21 (0.51, 2.91)	1.11 (0.67, 1.83)
Age- and race-adjusted OR (95% CI)	reference	1.23 (0.51, 2.97)	1.06 (0.63, 1.76)
Weak urinary stream symptom score			
≤1	1,265 (62.65)	24 (55.81)	93 (65.96)
>1	754 (37.35)	19 (44.19)	48 (34.04)
Unadjusted OR (95% CI)	reference	1.33 (0.72, 2.44)	0.87 (0.60, 1.24)
Age- and race-adjusted OR (95% CI)	reference	1.43 (0.76, 2.69)	0.79 (0.54, 1.15)

* Percents based on non-missing values

Table 4

Change in BPH/LUTS characteristics over time by diabetes status in community-dwelling white and black men at baseline

Characteristic	No Diabetes (n=1,290) Median (Q1, Q3)	Diabetes (no meds) (n=23) Median (Q1, Q3)	Diabetes (meds) (n=78) Median (Q1, Q3)	p-value‡
Annual Change				
AUASI score (points)	0.00 (−0.25, 0.56)	0.00 (−0.91, 0.94)	0.00 (−0.91, 1.31)	0.89
Prostate volume (%)†	4.39 (0.22, 8.26)	3.54 (1.24, 8.76)	4.70 (1.83, 9.03)	0.87
Maximum urinary flow rate (%)†	−2.16 (−6.20, 2.10)	−4.20 (−6.25, 3.55)	−1.66 (−3.20, 5.86)	0.53
Total PSA (%)†	4.90 (−3.04, 13.67)	3.35 (−1.31, 11.26)	3.41 (−10.21, 7.64)	0.57

†Measures on men with in-clinic examinations only: 364 No Diabetes, 14 Diabetes (no meds), and 28 Diabetes (meds))

‡Kruskal-Wallis p-value for test for differences across diabetes categories