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Influence of Vascular Comorbidities and Race on Erectile Dysfunction after Prostate Cancer Radiotherapy

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Abstract

Introduction—Vascular comorbidities (VC) (hypertension, diabetes, and hyperlipidemia) are known factors related to erectile dysfunction (ED) in men. However, no data are yet available for the effects of VC on ED incidence after prostate cancer radiotherapy (XRT).

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Aim—To investigate the influence of VC on post-XRT ED incidence and to further characterize ED incidence by racial groups.

Main Outcome Measures—ED incidence.

Methods—We reviewed 732 charts of patients (267 Caucasian and 465 African American [AA]) who received prostate XRT (external beam radiotherapy and/or brachytherapy) with or without hormone therapy between 1999 and 2010. The number of pre-XRT VC (0, 1, 2, or 3) was determined by medical history and medication list. ED (defined by use of erectile aids or by documentation of moderate or high sexual dysfunction on patient history) was determined pre-XRT as well as 1, 2, and 4 years post-XRT.

Results—ED incidence progressively increased from 22% pre-XRT to 58% 4 years post-XRT ($P < 0.01$). Additionally, ED incidence significantly increased with number of VC—4-year incidence between patients with 1 vs. 0 ($P = 0.02$), 2 vs. 0 ($P < 0.01$), 3 vs. 0 ($P < 0.01$), 3 vs. 1 ($P < 0.01$), and 3 vs. 2 ($P = 0.04$) VC (2 vs. 1 VC was nonsignificant). Compared with the Caucasian patients, ED incidences were slightly higher for the AA group with 0, 1, 2, and 3 comorbidities at 4 years follow-up (but statistically nonsignificant).

Conclusions—The number of VCs have a significant effect on development of post-XRT ED. Pre- and post-XRT ED appear to be independent of race when number of VCs are considered. Our results can be used to guide physicians in counseling patients on the incidence of ED by number of VC and as preliminary data for prospective efforts aimed at reducing post-XRT ED.

Keywords

Prostate Cancer; Radiotherapy; Erectile Dysfunction; Vascular Comorbidity; Race

Introduction

With advances in diagnosis and treatment, over 2.35 million American men have currently survived prostate cancer [1]. As such, reduction of treatment-related side effects is increasingly important. Erectile dysfunction (ED) is a common complication of prostate cancer radiotherapy (XRT) [2,3]. Depending on the population studied, up to 50% of men receiving radiotherapy for prostate cancer will suffer post-XRT ED [4]. Clearly, radiation-induced ED is a clinical problem of enormous magnitude.

The causes of radiation-induced ED are not fully understood, with several different proposed factors and mechanisms, including neurogenic, vascular, and psychogenic [5]. It has been proposed that dose-related vascular damage to the neurovascular bundles may be a critical causative factor [3]. Several studies have been conducted to understand the radiation dose to penile structures (body and bulb) and radiation-induced ED [6,7].

Hypertension (HTN), diabetes (DM), and hyperlipidemia (HL) are the three most common vascular comorbidities (VC) associated with ED in the general population, most likely due to the common mechanism of vasculogenic damage [8]. However, the influence of these VC on post-XRT ED is poorly understood. The prevalence of HTN and DM is higher in African Americans (AAs) than Caucasians, and this has been found to translate to an overall higher

age-matched prevalence of ED in AA men [9]; the higher prevalence of VC has in some cases served as an obstacle to enrollment of AA men on quality of life trials.

While vascular disease is clearly related to ED, little is known about the effect of XRT on ED, and no published reports to date exist on the effect of vascular disease on post-XRT ED. Therefore, we undertook this study to understand the impact of VC on post-XRT ED and to further characterize pre- and post-XRT ED by race.

Materials and Methods

After obtaining institutional review board approval, the charts of consecutive prostate cancer patients treated with radiation therapy at two affiliated hospitals in our department between 1999 and 2010 were reviewed. Patients with stage IV disease, patients who had radical prostatectomy, and patients with follow-up data less than 1 year were excluded. The remaining 732 patients (267 Caucasians and 465 AAs) comprise the study population. Our standard clinical practice is to have providers evaluate ED at each visit with questions from the provider. Patients were asked if they were having problems with obtaining erections adequate enough for sexual activity and were charted on a three-tiered system for having (i) no or minimal sexual dysfunction, (ii) moderate sexual dysfunction, or (iii) severe sexual dysfunction. Patients reporting moderate or severe problems with erectile function were referred for urologic evaluation and treatment of ED.

The medical records were used to abstract demographic, disease, and treatment information. Patient's VC (HTN, DM, and HL) status was determined by medications and past medical history recorded in patient chart during patients' initial consultation visit. Sexual function status was also determined by medical record review. For our study, ED was defined as a dichotomous event occurring if (i) moderate or severe sexual dysfunction was documented in patient chart on physician-obtained history or (ii) if patients were using or prescribed any erectile aids that included phosphodiesterase type 5 inhibitors, transurethral prostaglandin, intracavernosal injection therapy, or vacuum erection device [10]. Of note, patients who reported diminished erections but were still firm enough for sexual activity were considered as potent. Sexual function status was determined at four specific time points: pre-XRT, and 1 year, 2 years, and 4 years post-XRT. The median follow-up for all patients is 33.6 months. Intention-to-treat analysis was applied; for those patients lost to follow-up, their sexual function status was determined at the last appointment.

External beam radiotherapy (EBRT) was performed using three-dimensional conformal or intensity-modulated technique. If EBRT was given alone (i.e., without brachytherapy [BT]), the total dose to the prostate was 67.0 to 81.0 Gy delivered in 1.8 to 2.0 Gy fractions. Planning target volume (PTV) included the prostate, seminal vesicles, and in some cases pelvic lymph nodes to define PTV1; PTV2 consisted of prostate alone. BT was a component of XRT for 190 patients. When BT was combined with EBRT, and dose of EBRT was 45.0 Gy delivered in 25 fractions to the prostate plus seminal vesicles; EBRT PTV margins in all cases were typically 0.8–1.0 cm circumferentially with a smaller margin (0.6–0.8 cm) posteriorly. The BT dose was 100–109 Gy when combined with EBRT and 125–145 Gy when given alone.

Statistical analyses were performed using SAS (version 9.3, SAS Institute Inc., Cary, NC, USA) and Minitab (version 16, Minitab Inc., State College, PA, USA). The age difference between Caucasians and AAs was tested using the Student's *t*-test. The chi-square independence test was applied to determine the difference in demographic, disease, and treatment factors based on race. In order to determine the impact of comorbidity on radiation-induced ED, the two sample proportion test was performed to assess the significance of ED differences between patients with different number of VC. A logistic regression model was used to analyze the effect of race on radiation-induced ED by number of VC. All *P* values reported are two-tailed, with *P* < 0.05 considered statistically significant.

Results

Mean follow-up for the entire cohort was 33.6 months (Caucasian: 38.4 months, AA: 30.0 months). The clinical characteristics of the study population are shown in Table 1. As shown, AA and Caucasian patients were generally balanced with respect to Gleason score, radiation treatment, and use of hormone therapy. However, the AA patients were on average approximately 5 years younger, had a higher pretreatment prostate-specific antigen (PSA), had a lower T-stage, and had higher incidence of HTN (80% vs. 71%, *P* < 0.01) and DM (24% vs. 14%, *P* < 0.01) than Caucasian patients. However, the HL incidence for AA patients was lower than for Caucasian patients (36% vs. 44%, *P* < 0.01). Notably, the HTN, DM, and HL incidence differences between two racial groups identified in our study was consistent with reported results in general population [11].

In order to investigate the impact of VC on ED incidence post radiotherapy, we identified each patient's HTN, DM, and HL status prior to radiation and stratified them into four subgroups based on the number of VC (0, 1, 2, and 3). Table 2 displays the number/percentage of patients by number of VC across race groups. Overall there was a similar incidence of patients having 0, 1, or 2 VC, with the AA group having a higher percentage of patients with 3 VC.

Table 3a displays the incidence of ED as a function of number of VC and time. As shown, after radiotherapy, ED incidence increased in two manners: First, compared with the baseline levels (pre-XRT), ED incidence progressively increased from 1 year to 4 years post-XRT when patients had the same number of VC. Second, post-XRT ED incidence increased with increasing of number of comorbidities. Table 3b shows the results of pairwise comparisons of ED incidence by number of VC—these pairwise comparisons were done at two time points (pre-XRT and 4 years post-XRT). At 4 years post-XRT, the increases in ED incidence for all pairwise comparisons were statistically significant except the 2 vs. 1 comparison.

The analysis of ED incidence by race is shown in Table 4—Table 4a shows the comparisons by race at the 4 time points—pre-XRT and 1, 2, and 4 years post-XRT. As shown, although there was a slightly higher ED incidence in AA men at all time points (due perhaps to the slightly higher VC incidence in AA men), these differences in ED rates did not reach statistical significance. Table 4b displays the results of the logistic regression analysis of ED

incidence at each of the 4 time points by number of VC. None of these regression analyses at any time point demonstrated significant differences by race.

Figure 1 comprehensively displays the general results shown in Tables 3 and 4 visually. As shown, ED incidence steadily rises with time interval post-XRT and with increasing number of VC, with only a slight difference by race within each interval/VC-based bin.

Discussion

Our study is the first to report pre- and post-XRT ED as a function of VCs and race. HTN, DM, and HL are well-known risk factors for ED in the general population [8], which was consistent with our finding that, prior to radiation treatment, ED incidence increased with increasing number of VC (Table 3). In the post-XRT setting, we found a progressive increase in ED with increasingly longer post-XRT time interval, regardless of number of VC. At 1-year follow-up, the overall ED incidence for all patients was 52%, which is similar to other series [12,13]. An important and novel finding in our study was that the number of VC had a profound impact on development of radiation-induced ED. Indeed, at all three post-XRT analysis time points (Table 3 and Figure 1), the post-XRT ED incidence increased substantially with increasing the number of VC. Also, as shown in Table 3, the majority of the pairwise ED incidence comparisons based on number of VC reached statistical significance. Furthermore, when the number of VC within each comparison differed by 2 or more (i.e., 3 vs. 0, 3 vs. 1, and 2 vs. 0), the differences in ED incidence were highly significant, further highlighting that number of VC plays a critical role in increasing the rate of post-XRT ED.

Our study results clearly demonstrate that radiation-induced ED incidence increases when patients have more VCs (HTN, HL, and DM). It will be quite interesting to understand the underlying mechanism. Our results may support the hypothesis that post-XRT ED is due to vasculogenic insufficiency [5]. Several studies have demonstrated (by Doppler ultrasound) a reduction of blood flow in the cavernosal artery in men with ED after radiotherapy [14,15]. It has been suggested that radiotherapy causes pathological changes in small- and medium-sized arteries, which decrease vascular inflow. These arterial changes resemble chronic, progressive atherosclerosis and may be due to a combination of intimal fibrosis, direct damage to the arterial wall, and acceleration of naturally occurring atherosclerosis [16]. The first animal study demonstrated fibrosis change in the arteries of rat corpora cavernosa after adequate irradiation [17]. In addition to vascular parameters, XRT may have a direct effect on endothelial cell function, which is an area of strong interest in ED research [18]. Data from other body sites indicates that endothelial cells may be the initial target for damage during radiotherapy [19]. Perhaps radiation accelerates the process of atherosclerosis particularly if the patient has one or more VCs. This model is consistent with the findings of the present study, namely that post-XRT ED incidence increased when patients had more VCs.

Interventions that seek to minimize vascular damage during RT may be a reasonable strategy. Radiation Therapy Oncology Group (RTOG) 08-31 is a randomized, placebo-controlled trial currently examining the role of tadalafil for prophylaxis against XRT-

induced ED [20]. As a follow-up to this study, perhaps future studies examining interventions should control for number and severity of VC.

Our study is also the first to analyze pre- and post-XRT ED incidence by race. Pre-XRT ED incidence was slightly higher in AA (Table 4), likely due to the higher number of VC. This finding is consistent with other studies. Our study further shows, though, that when accounting for VC, there are no significant differences in ED incidence as a function of race. This is an important finding and will facilitate further studies that can focus on the impact of VC (particularly DM and HTN) on patient selection for prostate cancer quality of life trials and to enhance enrollment of AA patients on these trials.

There are several limitations to our study. First, we did not assess severity of the VCs or whether they were controlled through medication or diet—this will be done in a future analysis. Second, the definition of ED for this study was dichotomous and was based on abstraction of medical record (history of ED documented on history or follow-up or use of medications/treatment aids for ED). The use of validated instruments [21] would certainly increase our ability to accurately assess sexual function. Indeed, in a subset analysis of the Massachusetts Male Aging Study, a single-question self-report was found to be able to identify ED as well as a urologic examination including a sexual history, medical history, psychosocial history, and physical exam [22]. However, it should be noted that our definition of ED was applied uniformly to all patients across subsets so any potential recruitment biases based solely on the ED definition would be minimal. Third, though the Caucasian and AA populations were balanced overall, unbalanced items (such as age and PSA) may be confounding factors; it should be noted, however, that no significant differences based on race were demonstrated in any of the analyses based on time points or number of VC. Fourth, hormone treatment is a well-known factor to cause ED. Therefore, we investigated the hormone treatment rate among patients with different number of VC. As shown in Table 5, the hormone treatment percentage is not increasing as the number of VC increases. Fifth, the ED rates may vary as a function of radiation modality (EBRT alone vs. combination of EBRT with BT) [4,23], due in part perhaps to differences in radiobiological doses between these modalities [24]. However, results as a function of radiation modality (EBRT + BT) are shown in Table 6—as shown, the results seem to be consistent for patients with different numbers of VCs. Sixth, the dose to the penile structures may be another confounding factor because it has been shown to correlate with radiation-induced ED [6,25]. This is an ongoing area of investigation that we plan to analyze and report separately at a later date. Seventh, as our study population was in a single hospital system treated over a time period in which older radiotherapy techniques were used for some patients, our work requires validation in a larger study population treated with intensity-modulated radiotherapy, perhaps in the multi-institutional consortium setting [26,27]. Finally, the biases inherent to all retrospective analyses are understood by the investigators.

Despite these limitations, our study provides evidence that VC play an important role in the development of both pre- and post-XRT ED. Our results can be used to guide physicians in counseling patients on the incidence of ED by number of VC. Our results can also serve as preliminary data for prospective efforts aimed at reducing post-XRT ED and for future

investigations focusing on the incorporation of VC into ED-related treatment management decisions.

Conclusions

Our analysis represents the first effort in exploring post-XRT ED by VC and race. Our results suggest that number of VC have a significant effect on the development of post-XRT ED. Pre- and post-XRT ED appear to be independent of race when 1, 2, or 3 VC are considered. These findings can be useful for physicians in patient's consultation regarding post-XRT ED and as preliminary data for prospective efforts aimed at reducing post-XRT ED.

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Figure 1.

Pre-RT and post-RT ED incidences. At each time point, the ED incidences for each subgroup of patients with 0 (blue), 1 (red), 2 (yellow), and 3 (purple) vascular comorbidities are shown. For each time point, Caucasian patients are presented on the left and AA patients on the right.

Table 1

Patient characteristics

	Caucasian (total: 267)	AA (total: 465)	P value (<i>t</i> -test or chi-square)
Comorbidity			
HTN*	190 (71%)	372 (80%)	< 0.01
DM*	37 (14%)	112 (24%)	< 0.01
HL*	177 (44%)	167 (36%)	< 0.01
Years (mean)	68.6 ± 8.05	63.4 ± 8.10	< 0.01
Stage			
I	147 (55%)	324 (69%)	< 0.01
II	102 (38%)	107 (23%)	
III	15 (6%)	31 (7%)	
Unknown	3 (1%)	3 (1%)	
PSA (ng/ml)			
< 10	191 (74%)	256 (55%)	<0.01
10	76 (26%)	209 (45%)	
Gleason score			
Average	7.0 ± 0.88	7.0 ± 0.87	0.91
4–6	80 (30%)	147 (32%)	
7	130 (49%)	228 (49%)	
8–10	52 (21%)	88 (19%)	
Unknown	1 (<1%)	2 (<1%)	
Radiation treatment			
EBRT alone	192 (72%)	350 (75%)	0.10
BT ± EBRT	79 (28%)	116 (25%)	
Hormone therapy			
LHRH alone	78 (29%)	141 (30%)	0.35
LHRH/Antiandrogen	40 (15%)	100 (22%)	
Antiandrogen alone	3 (1%)	2 (<1%)	
None	146 (55%)	222 (48%)	

* Patient may or may not have other comorbidities.

Table 2

Prevalence of vascular comorbidities for Caucasian and AA prostate cancer patients

	Caucasian (total: 267)	AA (total: 465)	<i>P</i> value [*]
Patients without comorbidity			
None	50 (19%)	67 (14%)	0.16
Patients with one comorbidity			
HTN	82 (31%)	179 (39%)	0.03
DM	2 (1%)	7 (2%)	0.33
HL	25 (10%)	15 (3%)	<0.01
Total	109 (41%)	201 (43%)	0.53
Patients with two comorbidities			
HTN and DM	16 (6%)	45 (10%)	0.06
HTN and HL	73 (27%)	92 (20%)	0.02
DM and HL	1 (<1%)	5 (1%)	N/A
Total	90 (34%)	142 (31%)	0.38
Patients with three comorbidities			
HTN, DM, and HL	18 (7%)	55 (12%)	0.02

* Obtained using *t*-test.

Table 3

Erectile dysfunction incidences by vascular comorbidities (VC)

a. ED incidence for whole group by VC and time point

Number of VCs	Pre-XRT ED (%)	Post-XRT ED (%)		
		1 year	2 years	4 years
0	15	39	41	44
1	19	48	55	56
2	26	55	59	63
3	35	71	73	75
Overall	22	52	56	58

b. Pairwise comparisons of increase in ED incidence by number of VC

VC	Pre-XRT		4 years post-XRT	
	Increase in ED incidence (%)	<i>P</i> value*	Increase in ED incidence (%)	<i>P</i> value*
1 vs. 0	4	0.41	12	0.02
2 vs. 1	7	0.08	7	0.12
3 vs. 2	9	0.09	12	0.04
2 vs. 0	11	0.03	19	<0.01
3 vs. 1	16	<0.01	19	<0.01
3 vs. 0	20	<0.01	31	<0.01

* Obtained using *t*-test.

Table 4

Erectile dysfunction incidence by race

a. Overall pre- and post-XRT ED incidences for Caucasian and AA patients

	Caucasian (%)	AA (%)	P value*
Pre-XRT ED	20	23	0.31
Post-XRT ED			
1 year	48	54	0.12
2 years	52	58	0.12
4 years	55	60	0.21

b. Logistic regression analyses of ED differences by time point, number of VC, and race

Number of VCs	Post-XRT ED							
	Pre-XRT ED				1 year			
	Caucasian	AA	Caucasian	AA	Caucasian	AA	Caucasian	AA
0	14%	16%	38%	40%	38%	43%	40%	46%
1	18%	19%	46%	49%	52%	56%	55%	57%
2	22%	28%	52%	57%	56%	61%	60%	64%
3	33%	36%	67%	73%	67%	75%	71%	76%
P value [†]	0.37				0.53			
					0.30			

* Obtained using t-test

[†] Obtained using chi-square test.

Table 5

Hormone therapy use among patients by number of vascular comorbidities

	Caucasian (total: 267)	AA (total: 465)	<i>P</i> value *
Patients without comorbidity	50	67	
Hormone therapy	27 (54%)	39 (58%)	0.66
Patients with one comorbidity	109	201	
Hormone therapy	49 (45%)	113 (56%)	0.06
Patients with two comorbidities	90	142	
Hormone therapy	37 (41%)	65 (46%)	0.46
Patients with three comorbidities	18	55	
Hormone therapy	7 (39%)	24 (44%)	0.71

* Obtained by *t*-test.

Table 6

Brachytherapy use among patients by number of vascular comorbidities

	Caucasian (total: 267)	AA (total: 465)	<i>P</i> value *
Patients without comorbidity	50	67	
BT ± EBRT	16 (32%)	19 (28%)	0.69
Patients with one comorbidity	109	201	
BT ± EBRT	27 (25%)	42 (21%)	0.48
Patients with two comorbidities	90	142	
BT ± EBRT	23 (26%)	40 (28%)	0.76
Patients with three comorbidities	18	55	
BT ± EBRT	5 (28%)	12 (22%)	0.75

* Obtained by *t*-test.

BT = brachytherapy; EBRT = external beam radiotherapy