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Original Article

Is Computed Tomography a Necessary Part of a Metastatic Evaluation for Castration-Resistant Prostate Cancer? Results From the Shared Equal Access Regional Cancer Hospital Database

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BACKGROUND: Metastatic lesions in prostate cancer beyond the bone have prognostic importance and affect clinical therapeutic decisions. Few data exist regarding the prevalence of soft-tissue metastases at the initial diagnosis of metastatic castration-resistant prostate cancer (mCRPC). **METHODS:** This study analyzed 232 men with nonmetastatic (M0) castration-resistant prostate cancer (CRPC) who developed metastases detected by a bone scan or computed tomography (CT). All bone scans and CT scans within the 30 days before or after the mCRPC diagnosis were reviewed. The rate of soft-tissue metastases among those undergoing CT was determined. Then, predictors of soft-tissue metastases and visceral and lymph node metastases were identified. **RESULTS:** Compared with men undergoing CT (n = 118), men undergoing only bone scans (n = 114) were more likely to have received primary treatment ($P = .048$), were older ($P = .013$), and less recently developed metastases ($P = .018$). Among those undergoing CT, 52 (44%) had soft-tissue metastases, including 20 visceral metastases (17%) and 41 lymph node metastases (35%), whereas 30% had no bone involvement. In a univariable analysis, only prostate-specific antigen (PSA) predicted soft-tissue metastases (odds ratio [OR], 1.27; $P = .047$), and no statistically significant predictors of visceral metastases were found. A higher PSA level was associated with an increased risk of lymph node metastases (OR, 1.38; $P = .014$), whereas receiving primary treatment was associated with decreased risk (OR, 0.36; $P = .015$). **CONCLUSIONS:** The data suggest that there is a relatively high rate of soft-tissue metastasis (44%) among CRPC patients undergoing CT at the initial diagnosis of metastases, including some men with no bone involvement. Therefore, forgoing CT during a metastatic evaluation may lead to an underdiagnosis of soft-tissue metastases and an underdiagnosis of metastases in general. ***Cancer* 2016;122:222-9.** *© 2015 American Cancer Society.*

KEYWORDS: castration-resistant prostatic neoplasms, computed X-ray tomography metastasis, logistic models, prevalence, prostate-specific antigen, soft-tissue neoplasms.

INTRODUCTION

Because bone is the most common site for prostate cancer metastases, bone scans play a central role in a prostate cancer metastatic evaluation.[1] Historically, the presence of metastases outside the bone (ie, soft tissue), including metastases of the visceral organs and distant lymph nodes, was thought to be rare. Recently, there has been a growing awareness of the importance of visceral metastases. This in part stems from the use of new life-prolonging therapies (eg, abiraterone, enzalutamide, and docetaxel) that may alter the biology of prostate cancer and result in increased rates of visceral metastases.[2,3] The detection of visceral metastases as well as distant nodal metastases has 3 important clinical implications. First, the median survival of patients with visceral metastases or a combination of nodal metastases and bone metastases is significantly

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shorter than that of patients with only bone metastases.[4] Second, clinical trials of newer prostate cancer therapies have excluded men with visceral metastases (eg, radium-223, sipuleucel-T, and abiraterone in men with chemotherapy-naive castration-resistant prostate cancer [CRPC]) or nodal metastases greater than 3 cm (eg, radium-223) because of their poorer prognosis in general. As such, the effectiveness of these agents in this group of men is unknown. In contrast, trials of other drugs (eg, enzalutamide, cabazitaxel, cabozantinib, and abiraterone in men after docetaxel) included these men and showed a benefit in subgroup analyses for men with visceral metastases.[5-10] Third, visceral and lymph node metastases may lead to a ureteral obstruction that requires intervention. Knowledge of soft-tissues metastases may not only affect the prognosis but also influence treatment decisions.

Although the clinical implications are becoming increasingly clear, the exact prevalence of soft-tissue or visceral metastases is unclear. Estimates of the prevalence of soft-tissue metastases range from 35% to 45% in phase 3 clinical trials of docetaxel and ketoconazole for metastatic castration-resistant prostate cancer (mCRPC) to 50% in trials of men who progressed after docetaxel to 100% in a small autopsy study of 30 men who died from prostate cancer.[5-7,11-15] Estimates of visceral metastases in these settings range from 22%-24% to 23%-29% to more than 66%, respectively.[5-7,11-15] The estimated prevalence of nodal metastases ranges from 31% in the trial of ketoconazole to 63% in the autopsy study.[12,14] However, the prevalence of all types of soft-tissue metastases at the initial diagnosis of mCRPC is unclear.

To fill this gap in the literature, we examined the prevalence of soft-tissue metastases at the time of the initial diagnosis of mCRPC among patients who underwent computed tomography (CT) imaging with or without bone imaging. We then examined predictors of any soft-tissue metastases, visceral metastases, and lymph node metastases at the time of the initial mCRPC diagnosis to assess the importance of CT scanning for the diagnosis of distant prostate cancer metastases.

MATERIALS AND METHODS

Data Collection

After obtaining institutional review board approval, we identified 668 patients who were being treated at the Veterans Affairs hospital in Durham, NC or San Diego, Calif. between 2000 and 2013 and developed M0 CRPC. CRPC was defined in accordance with the Prostate-Specific Antigen (PSA) Working Group 2 criteria: a $\geq$ 25% PSA increase and an absolute $\geq$2 ng/mL increase

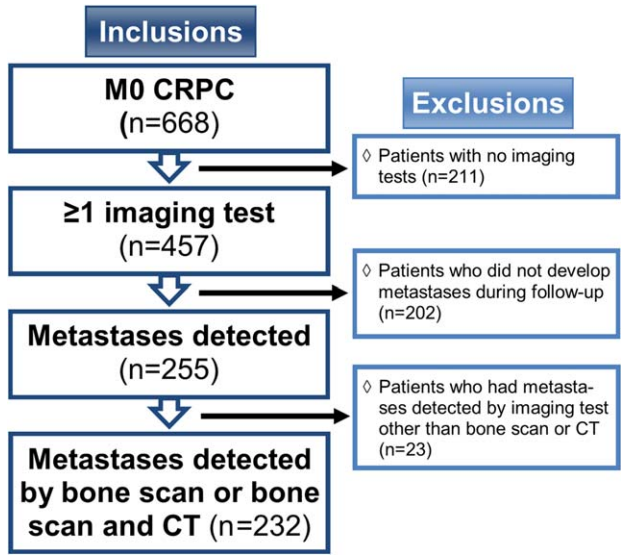

Figure 1. Consolidated Standards of Reporting Trials diagram for patients. CRPC indicates castration-resistant prostate cancer; CT, computed tomography.

from the post–androgen -deprivation therapy nadir while the patient is castrate.[16] Patients with metastases before CRPC were not included to limit the study to M0 CRPC patients. We defined castration as a testosterone level < 50 ng/dL, bilateral orchiectomy, or the continuous receipt of a luteinizing hormone–releasing hormone agonist or antagonist. Detailed methods on the selection of our population have been published previously.[17] We collected information on demographic, clinical, and pathological characteristics as well as all imaging after the CRPC diagnosis. Of the 668 men with M0 CRPC, 457 (68%) had at least 1 imaging test after the diagnosis of M0 CRPC, and 255 of these men (56%) had a positive imaging test for metastases (Fig. 1). Data were collected from any bone scan or CT scan within the 30 days before or after the initial metastasis diagnosis. Imaging tests were coded by trained personnel as positive or negative for bone metastases or soft-tissue metastases on the basis of the radiology report (equivocal scans, because they usually do not prompt a change in management, were considered negative unless they were confirmed positive by biopsy). CT scans were evaluated solely for the presence of soft-tissue metastases (metastatic sites other than bone). Lymph nodes were considered metastatic if they were outside the pelvic region and greater than or equal to 2 cm. We considered visceral metastases to be any soft-tissue metastases excluding lymph nodes; they included the liver, lungs, peritoneal carcinomatosis, colon, and brain. We excluded patients who were diagnosed with metastases

TABLE 1. Baseline Characteristics at the Time of Metastases

	Overall (n = 232)	Bone Scan Only (n = 114)	CT ± Bone Scan (n = 118)	*P*
Age, median (IQR), y	75 (67-81)	77 (71-81)	73 (65-81)	.013
Year of metastases, median (IQR)	2006 (2004-2010)	2006 (2004-2009)	2007 (2005-2010)	.018
Race, No. (%)				.923
Nonblack	156 (67)	77 (68)	79 (67)	
Black	76 (33)	37 (32)	39 (33)	
Center, No. (%)				.192
1	120 (52)	54 (47)	66 (56)	
2	112 (48)	60 (53)	52 (44)	
Primary localized treatment, No. (%)				
None	83 (36)	35 (30)	48 (42)	.048
Radical prostatectomy ± radiation	149 (64)	83 (70)	66 (58)	
PSA at metastases, median (IQR), ng/mL	48.9 (16.2-131.1)	45.7 (13.8-144.1)	50.0 (19.2-111.9)	.914
PSADT at metastases, No. (%)				.284
≥9 mo	51 (22)	24 (21)	27 (23)	
3-8.9 mo	93 (40)	46 (40)	47 (40)	
<3 mo	43 (19)	17 (15)	26 (22)	
Missing	45 (19)	27 (24)	18 (15)	
Soft-tissue metastases, No. (%)[a]			52 (44)	
Lymph node metastases	—	—	41 (35)	
Visceral metastases			20 (17)	
Liver	—	—	11 (9)	
Lung	—	—	3 (3)	
Other visceral	—	—	10 (8)	
Months from CRPC to metastases, median (IQR)	14.2 (5.4-30.9)	13.9 (4.5-31.7)	14.7 (6.1-28.9)	.910

Abbreviations: CRPC, castration-resistant prostate cancer; CT, computed tomography; PSA, prostate-specific antigen; PSADT, prostate-specific antigen doubling time; IQR, interquartile range.
[a] The count of total soft-tissue metastases does not match the sum of the individual subcategories because patients could have multiple types of metastases.

by an imaging test other than a bone scan or CT (n = 23), and this resulted in a final study population of 232 men with CRPC who had metastases first detected by a bone scan or CT. Imaging tests were ordered at the discretion of the treating physician.

Statistical Analysis

The prostate-specific antigen doubling time (PSADT) was calculated by the division of the natural logarithm of 2 (0.693) by the slope of the linear regression of the natural logarithm of PSA over time in months. PSADT was calculated with all available PSA levels in the 2 years leading up to metastases or with PSA levels starting at the time of the CRPC diagnosis if metastases were detected within 2 years of the CRPC diagnosis. Because there were insufficient PSA data to calculate PSADT for 45 patients (19%), we categorized PSADT into 3 groups (0-2.9, 3-8.9, and ≥9 months) on the basis of previous literature[18] and included a fourth group of those missing PSADT data.

Patients were classified into 2 groups: those who underwent a bone scan only during their metastatic evaluation and those who underwent CT with or without a bone scan during their metastatic evaluation. Characteristics were compared between patients in these 2 groups with chi-square tests for categorical variables and with rank-sum tests for continuous variables.

A logistic model for predicting the presence of soft-tissue metastases was fit among those who underwent CT with or without a bone scan. We first fit univariable logistic models to examine the association between each variable of interest and the risk of soft-tissue metastases. Then, we used forward selection with an entry criteria of $\alpha = .05$ to select variables for the model. Predictors that were considered included age (continuous), year of metastasis (continuous), race (black vs nonblack), treatment center, primary localized treatment (none vs radical prostatectomy and/or radiation), PSA at the time of metastasis (continuous and log-transformed), PSADT leading up to metastases (0-2.9 months vs 3-8.9 months vs ≥ 9 months vs missing), and months from the CRPC diagnosis to metastases (continuous). The accuracy of the model was assessed with the area under the curve.

In the secondary analysis, we used univariable logistic regression to examine the association between the risk of visceral metastases and the risk of lymph node metastases with the predictors listed previously among patients who underwent a CT scan. Because of the small numbers, we were unable to perform a multivariable analysis.

RESULTS

Of the 232 men who had M0 CRPC and then developed metastases, 114 (49%) had a bone scan only, and 118

TABLE 2. Predictors of Soft-Tissue Metastases

	Univariable Analysis			Model Selection[a]		
	OR	95% CI	*P*	OR	95% CI	*P*
Age	1.01	0.97-1.05	.585	—		
Race						
Nonblack	Reference			Reference		
Black	0.83	0.38-1.81	.640	—		
Year	0.96	0.87-1.07	.451	—		
Center						
1	Reference			Reference		
2	1.54	0.74-3.21	.250	—		
Primary localized treatment						
None	Reference			Reference		
Radical prostatectomy ± radiation	0.47	0.21-1.05	.066	—		
PSA at metastases[b]	1.27	1.00-1.60	.047	1.27	1.00-1.60	.047
PSADT at metastases						
≥9 mo	Reference			Reference		
3-8.9 mo	1.76	0.66-4.71	.260	—		
<3 mo	2.73	0.89-8.33	.078	—		
Missing	1.00	0.28-3.54	.999	—		
Months from CRPC to metastases	0.99	0.98-1.01	.412	—		

Abbreviations: CI, confidence interval; CRPC, castration-resistant prostate cancer; OR, odds ratio; PSA, prostate-specific antigen; PSADT, prostate-specific antigen doubling time.
[a] Multivariable model using forward selection with an α value of .05 and an area under the curve of 0.623.
[b] Log-transformed.

TABLE 3. Type of Imaging Test and Type of Metastasis

	Bone scan only, No.	CT ± Bone Scan, No. (%)
Soft-tissue metastases	—	36 (30)
Bone metastases	Unknowable	66 (56)
Both soft-tissue and bone metastases	Unknowable	16 (14)
Total	114	118

Abbreviation: CT, computed tomography.

(51%) had CT with or without a bone scan (Table 1). Those who had a bone scan only were older (median age, 77 vs 73 years; $P = .013$), developed metastases less recently (median year, 2006 vs 2007; $P = .018$), and were more likely to receive primary localized treatment (70% vs 58%; $P = .048$) in comparison with those who underwent CT with or without a bone scan. There was no association between the scanning modality and race, treatment center, PSA at metastases, PSADT at metastases, or months from the CRPC diagnosis to metastases (all $P > .1$). The median PSA level before the diagnosis of mCRPC was 48.9 ng/dL for all patients, 45.7 ng/dL for patients undergoing only a bone scan, and 50.0 ng/dL for patients undergoing a CT scan with or without a bone scan. PSADT was greater than or equal to 9 months for 22% of the patients, 3 to 9 months for 40% of the patients, and less than 3 months for 19% of the patients, with 19% lacking sufficient PSA data for the calculation of PSADT. Of the patients who underwent CT and were diagnosed with metastases, 9% had liver metastases, 3% had lung metastases, 8% had other visceral metastases, 35% had lymph node metastases, and 30% had no bone involvement.

In the univariable analysis, only a higher PSA level at the time of metastases was statistically significant in predicting a higher risk of soft-tissue metastases among patients who underwent CT with or without a bone scan (odds ratio [OR], 1.27; 95% confidence interval [CI], 1.00-1.60; $P = .047$; Table 2). Indeed, men who had soft-tissue metastases had higher median PSA values (70.7 ng/mL) than men without them (43.9 ng/mL; $P = .038$). Although there was a trend between higher PSADT values and a higher risk of soft-tissue metastases, this did not reach statistical significance. Similarly, the trend between receiving localized primary treatment and a lower risk of soft-tissue metastases did not reach statistical significance. When forward selection was used, PSA was the only variable that entered the model (OR, 1.27; 95% CI, 1.00-1.60; $P = .047$). The area under the curve of this model was 0.623 (95% CI, 0.518-0.727). Among the patients who underwent CT with or without a bone scan, 66 (56%) had only bone metastases, 36 (30%) had soft-tissue metastases, and 16 (14%) had both soft-tissue and bone metastases (Table 3).

TABLE 4. Univariable Predictors of Visceral and Lymph Node Metastases

	Visceral Metastases			Lymph Node Metastases		
	OR	95% CI	*P*	OR	95% CI	*P*
Age	1.05	1.00-1.11	.062	1.00	0.96-1.04	.894
Race						
Nonblack	Reference			Reference		
Black	0.30	0.08-1.11	.071	1.08	0.48-2.41	.854
Year	1.01	0.88-1.15	.941	0.95	0.85-1.06	.351
Center						
1	Reference			Reference		
2	1.33	0.51-3.49	.628	1.56	0.73-3.34	.255
Primary localized treatment						
None	Reference			Reference		
Radical prostatectomy ± radiation	0.74	0.27-2.06	.567	0.36	0.16-0.82	.015
PSA at metastases[a]	0.95	0.71-1.27	.736	1.38	1.07-1.78	.014
PSADT at metastases						
≥9 mo	Reference			Reference		
3-8.9 mo	3.38	0.68-16.75	.136	1.13	0.42-3.07	.806
<3 mo	1.63	0.25-10.65	.610	2.00	0.66-6.07	.221
Missing	4.81	0.82-28.27	.082	0.25	0.05-1.33	.104
Months from CRPC to metastases	0.99	0.97-1.02	.659	1.00	0.98-1.02	.949

Abbreviations: CI, confidence interval; CRPC, castration-resistant prostate cancer; OR, odds ratio; PSA, prostate-specific antigen; PSADT, prostate-specific antigen doubling time.
[a] Log-transformed.

There were no significant predictors of visceral metastases. Although not statistically significant, there was a suggestion of an association between race and visceral metastases, with black men at lower risk for visceral metastases (OR, 0.30; 95% CI, 0.08-1.11; $P = .071$; Table 4). Although PSADT was not associated with visceral metastases, there was also a suggestion of an association between a missing PSADT value and a higher risk of visceral metastases (OR, 4.81; 95% CI, 0.82-28.27; $P = .082$). A higher PSA level at the time of metastases was associated with an increased risk of lymph node metastases (OR, 1.38; 95% CI, 1.07-1.78; $P = .014$), whereas receiving primary localized treatment was associated with a decreased risk of lymph node metastases (OR, 0.36; 95% CI, 0.16-0.82; $P = .015$). No other factors were associated with a risk of lymph node metastases.

DISCUSSION

Previous studies have shown that soft-tissue metastases correspond with worse outcomes than bone metastases alone.[4] Moreover, certain treatments for mCRPC are not indicated for men with soft-tissue metastases.[5,7-9] As such, determining the prevalence of soft-tissue metastases is clinically relevant. Despite this, few studies have estimated the prevalence of soft-tissue metastases at the time of the initial diagnosis of metastasis. In our retrospective analysis of men with CRPC and no prior metastases who all developed metastatic disease as detected on CT scans with or without bone scans, 44% had soft-tissue metastases at the time of the first metastasis, and this suggests that soft-tissue metastases are common at the time of the initial mCRPC diagnosis. In fact, a reasonable number of men had no bone involvement on a bone scan or did not undergo a bone scan at diagnosis; this suggests that if CT had not been performed, a portion of these men would have been considered nonmetastatic, although the precise number is unknown because we did not assess CT scans for possible bone metastases. We have no way of knowing how many soft-tissue metastases were missed among patients who underwent only a bone scan and not CT. Only 1 clinical variable (PSA) was a significant predictor of soft-tissue metastases, and its predictive accuracy was modest. In the absence of good clinical variables to select appropriate patients for imaging, our data suggest that all men should undergo body imaging for soft-tissue metastases. These results require validation in other data sets and the use of alternative body imaging modalities (ie, magnetic resonance imaging).

Historically, prostate cancer has been described as metastasizing primarily to the bone, with ≤10% of cases having soft-tissue metastases (with pelvic lymph nodes not being counted).[1] In recent years, mCRPC trials with protocol-mandated body imaging have identified a greater prevalence of soft-tissue metastases and noted worse outcomes for these patients.[5,7,11-15] Although many recent trials have excluded patients with visceral or nodal

metastases, others have included them and shown similar efficacy in these groups on subgroup analyses, albeit with a worse overall prognosis.[5,7-9] To date, our knowledge about the prevalence of soft-tissue metastases is derived either from these trials that allowed these patients on study or autopsy series.[5,7-9,11-15] However, these trials did not require the patient to be newly diagnosed with mCRPC. Thus, to our knowledge, no prior study has estimated the prevalence of soft-tissue metastases at the initial diagnosis of mCRPC, a critical juncture for treatment decisions. To evaluate the added benefit of using body imaging in a metastatic evaluation in addition to a bone scan, we reviewed our database of men with M0 CRPC who later developed metastases and determined the prevalence of soft-tissue metastases among men undergoing CT within the 30 days before or after the mCRPC diagnosis.

Among patients undergoing CT within 30 days of mCRPC, 44% had soft-tissue metastases. Specifically, 35% of the patients had distant lymph node metastases, and 17% had visceral metastases. Those with visceral metastases included 9% with liver metastases, 3% with lung metastases, and 8% with other types of visceral metastases, including colon, brain, and peritoneal carcinomatosis. These data are on par with previous estimates of soft-tissue metastases among mCRPC patients who have not received additional treatment; trials of docetaxel and ketoconazole for mCRPC reported a 35% to 45% prevalence of soft-tissue metastases, a 31% prevalence of nodal metastases, a 22% to 24% prevalence of visceral metastases, a 6% to 9% prevalence of liver metastases, and a 5% to 10% prevalence of lung metastases.[13-15] The overall prevalence of soft-tissue metastases that we found among men who underwent CT was consistent with the ranges found in the mCRPC trials of docetaxel and ketoconazole (45% vs 35%-45%), though on the high end in our study.[13-15] In comparison, patients who progressed after docetaxel (a later milestone in the natural history of prostate cancer) had a 50% prevalence of soft-tissue metastases.[5,7-9] The lower rates of visceral metastases found in our study of men who had undergone CT (17% vs 22%-24%) are consistent with expectations because our study's strict inclusion criteria counted only those metastases detected in the first month of diagnosis, whereas the docetaxel and ketoconazole studies included men with mCRPC regardless of when they were diagnosed with mCRPC.[13-15] Patients who progressed after docetaxel had a 23% to 29% prevalence of visceral metastases.[5,7-9] Finally, our study identified a similar prevalence of nodal metastases among those who had undergone CT in comparison with the mCRPC trial of ketoconazole (35% vs 31%).[14]

Using the same cohort of men featured in the current study, we previously evaluated the predictors of a positive bone scan. In that study, PSA was a very strong predictor of a positive bone scan with an OR of 2.11 ($P < .001$).[19] In comparison, in the current study, although PSA was significantly linked with soft-tissue metastases, the OR was much lower (1.27; $P = .047$). This suggests that although PSA is a robust predictor of bone metastases, it is not as helpful in identifying soft-tissue metastases. Likewise, PSADT was a very strong predictor of bone metastases in our prior study (OR, 0.53; $P < .001$) but was not a significant predictor of soft-tissue metastases in the current study.[19] Ultimately, the best model for predicting bone metastases in our prior study had an area under the curve of 0.773 (vs 0.623 in the current study) for predicting soft-tissue metastases.[19] Other studies have similarly shown that current tools are far from ideal in identifying soft-tissue metastases.[20-22] Collectively, these findings suggest that with currently available clinical tools, predicting soft-tissue metastases remains challenging.

The modest association between PSA and soft-tissue metastases in comparison with the strong association between PSA and bone metastases may be due, in part, to heterogeneity in the biology of soft-tissue metastases. In the secondary analysis, we found an association between PSA and lymph node metastases but not between PSA and visceral metastases, and this suggests that the association that we found between all types of soft-tissue metastases and PSA was primarily driven by the association between PSA and lymph node metastases. This observation was not unexpected because studies have associated visceral metastases with small cell and neuroendocrine phenotypes of prostate cancer, which produce little to no PSA.[2,3] Although the increasing evidence of different biological processes in visceral prostate cancer metastases has excluded these patients from some trials, further defining this mCRPC subgroup may open up alternative clinical trials targeted to this group (eg, platinum-based chemotherapy).

In our secondary analysis, we found that black men were less likely to have visceral metastases, although this finding did not reach statistical significance. Although this finding requires confirmation in larger studies, if confirmed, this might seem counterintuitive because black race is a strong predictor of more aggressive prostate cancer.[23] However, we speculate that there may be a possible explanation. Black men have been found to have greater concentrations of certain androgens,[24] which may accelerate the growth of typical androgen-driven prostate

cancers. If androgen-driven cancers behave more aggressively in black men, this could drive the association between black race and aggressive cancer while still allowing for a possibly reduced prevalence of small cell and neuroendocrine phenotypes of prostate cancer and associated visceral metastases. Interestingly, a prior article[25] showed that among men with M1 CRPC, black men tended to have better survival outcomes, although no difference was found in the rates of visceral metastases between black and white men. Nonetheless, our findings should be interpreted with caution because of the lack of statistical significance and small numbers; thus, these findings require validation in larger data sets.

Our analysis has several limitations. The study was retrospective in nature, and imaging tests were performed at the discretion of ordering physicians, so it is possible that the patients undergoing CT were at higher risk than the patients who did not undergo CT despite our failure to find significant differences between these 2 populations other than age, treatment year, and receipt of primary treatment. Because of the relatively small numbers of patients analyzed, a lack of sufficient statistical power may explain the failure to identify predictors of soft-tissue metastases other than PSA (especially PSADT). Although we found different associations in predictors of visceral and lymph node metastases, the numbers were modest, and the results must be interpreted with caution. Also, our data collection relied on several assumptions. The prevalence of metastases at the diagnosis of mCRPC was based on the assumption that imaging tests performed $\leq$30 days before or after the first metastasis were "at diagnosis"; tests performed afterward were considered to be follow-up scans. This was done to distinguish between metastases identified at diagnosis and those that developed with continued tumor progression (ie, during follow-up). Thus, delayed scans that were meant to be performed shortly after diagnosis but that actually occurred >30 days after the diagnosis of mCRPC were not counted; this possibly overestimated the frequency of not undergoing a CT scan at the diagnosis of mCRPC. Finally, the current study did not capture information about complications from soft-tissue metastases. However, because pelvic nodal disease, which was not captured in this study, can likewise lead to complications such as a ureteral obstruction, the current study may have underestimated the number of men at risk for complications from soft-tissue metastases.

In summary, this exploratory analysis found a 44% prevalence of soft-tissue metastases at the diagnosis of mCRPC among patients undergoing a CT scan with or without a bone scan as well as some men with no evidence of bone metastases, and it identified PSA as a modest predictor of soft-tissue metastases. Our data suggest that a CT scan should be routinely used for the metastatic evaluation of men with M0 CRPC to avoid missing soft-tissue metastases among men with mCRPC and to avoid missing patients who have only soft-tissue metastases and would otherwise be considered nonmetastatic.

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CONFLICT OF INTEREST DISCLOSURES

The authors made no disclosures.

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