Published in final edited form as:

Urology. 2015 February ; 85(2): 415–422. doi:10.1016/j.urology.2014.09.054.

Clinical Presentation and Outcomes of Prostate Cancer in an Urban Cohort of Predominantly African-American, HIV-infected Patients

David J. Riedel^{1,*a}, Eric R. Cox^{2,*}, Kristen A. Stafford³, and Bruce L. Gilliam¹

¹Institute of Human Virology and Division of Infectious Diseases, University of Maryland School of Medicine, Baltimore, MD

²JenCare Neighborhood Medical Center, Norfolk, VA

³Department of Epidemiology and Public Health, University of Maryland School of Medicine, Baltimore, MD

Abstract

Objective—To determine the clinical presentation and outcomes of prostate cancer in HIV-infected men compared to HIV-uninfected men in an urban setting.

Methods—A retrospective cohort study of prostate cancer stage at diagnosis and mortality comparing HIV-infected patients to HIV-uninfected patients from 2000 to 2011 was carried out. Clinical features, HIV history, cancer presentation, and outcomes were reviewed. Cox proportional hazards analysis was performed to estimate the association between HIV status and mortality.

Results—A total of 54 HIV-infected subjects were identified and reviewed and 49 had complete data available; they were compared to 1,496 HIV-uninfected subjects with prostate cancer. HIV-infected subjects were younger (median age 60.7 years vs. 64), and had a higher proportion of African-Americans (92% vs. 45%). Elevated PSA (76%) was the predominant indication for biopsy; ten (27%) patients with an elevated PSA had normal findings on digital rectal exam. Eighteen (37%) presented with stage III/IV disease, compared to 14% in the general population ($P < 0.001$). Eight patients (16%) died from prostate cancer. Subjects with HIV progressed to death at a significantly faster rate than those in the general population (adjusted hazard ratio 2.02, 95% CI 1.14–3.58).

© 2014 Elsevier Inc. All rights reserved.

^aCorresponding Author: David J. Riedel, MD, Institute of Human Virology and Division of Infectious Diseases, University of Maryland School of Medicine, Program in Oncology, University of Maryland Marlene and Stewart Greenebaum Cancer Center, 725 W. Lombard St., N552, Baltimore, MD, 21201, Tel: 410-706-5665, Fax: 410-706-4619, driedel@ihv.umaryland.edu.

*These authors contributed equally to this work

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

All others declare no conflicts of interest.

Author contributions: Study conception: DJR, ERC, BLG. Data acquisition: DJR, ERC. Data analysis and interpretation: DJR, ERC, KAS, BLG. Drafting the manuscript: DJR, ERC. Revisions: all. Approval of the final manuscript: all.

Conclusion—HIV-infected patients in this cohort presented with more advanced stage disease compared to the general population even though the majority were detected by screening PSA. The overall mortality rate was higher for HIV-infected patients with prostate cancer after controlling for race, tumor stage at diagnosis, and age. Prostate cancer screening methods may need to be individualized for HIV-infected men.

Keywords

HIV; AIDS; prostate cancer; PSA; non-AIDS-defining cancer; African-American

INTRODUCTION

HIV-infected patients are at increased risk for AIDS-defining and non-AIDS-defining cancers (NADCs). The incidence of most NADCs has been increasing over the last decade of the combined antiretroviral therapy (ART) era. Prostate cancer (categorized as a NADC) incidence in HIV-infected men from the early AIDS era was lower than in the general population.^{1–4} However, there remains some controversy, with several studies reporting higher rates.^{5–7} Lower rates have been attributed to less frequent prostate-specific antigen (PSA) screening in the HIV population⁸ and antineoplastic effects of certain antiretrovirals, specifically protease inhibitors (PIs).⁹ However, the widespread success of ART (and consequent increased survival of HIV-infected men) and the aging of the general HIV population are expected to lead to an increased rate of prostate cancer in the future.

Both HIV and prostate cancer disproportionately affect African-American men. African-American men are at significantly increased risk of developing prostate cancer and are more likely to present with advanced stage disease.¹⁰ Despite comprising a minority of the U.S. population, African-Americans account for nearly half of new HIV infections.¹¹ Several case reports^{12,13} have suggested that prostate cancer in HIV-infected patients may be more aggressive or present at more advanced stages than in the general population. Only small case series^{14–17} of HIV-infected patients with prostate cancer have been published to date, with conflicting information about stage of presentation and outcomes. One study recently reported that the risk of distant stage prostate cancer was elevated in the HIV-infected population in the U.S.¹⁸ Possible reasons for more advanced staging at diagnosis include biological differences in the tumors, impaired immune surveillance and cytotoxic killing, and reduced PSA screening in the HIV-infected population leading to delayed presentation.⁸

Although PSA testing has generally permitted earlier detection of prostate cancer, it is unclear whether this earlier detection translates into reduced mortality across all patient populations.¹⁹ The aim of this study was to determine the clinical characteristics, presentation, and outcomes of prostate cancer in HIV-infected men compared to the non-HIV-infected general population.

METHODS

To identify the exposed arm of our cohort, we reviewed the clinical records of all HIV-infected men diagnosed with prostate cancer in our facilities in Baltimore, Maryland from January 2000 to December 2011. The Institute of Human Virology provides comprehensive

HIV care to approximately 5,000 patients per year at University of Maryland Medical Center, Maryland General Hospital (now, University of Maryland Medical Center Midtown Campus), and Baltimore Veterans Affairs (VA) Medical Center. Data abstraction methods and analysis procedures have been previously described.²⁰ Exposed subjects were identified by searching electronic clinic records for diagnoses of HIV, AIDS, and prostate cancer. The University Clinical Data Repository was also searched using ICD9 codes for HIV or AIDS and for prostate cancer (185). At the VA, the clinical case registry of HIV-infected patients was searched by ICD9 code for prostate cancer.

Cancer diagnoses were confirmed by pathology and clinician reports. Demographic and related clinical data were abstracted from medical charts. HIV-specific data included HIV diagnosis date, RNA viral load, CD4 cell counts, and ART. HIV suppression was defined as any RNA viral load < 400 copies/ml (assays used over time had varying detection limits from 48–400 copies/ml). PSA values, DRE findings, time from abnormal PSA to biopsy, Gleason score, clinical stage, and all treatments were abstracted from the medical record. Subject status as of December 31, 2011 was recorded as alive, dead or unknown. Survival was calculated from the date of cancer diagnosis to death or conclusion of the follow up period, December 31, 2011. For comparison to the HIV-uninfected population, the University of Maryland Greenebaum Cancer Center tumor registry was searched for all cases of prostate cancer seen during the same time period, and available data included age, race, stage at diagnosis, and mortality outcomes.

Data were first investigated using univariate analysis to describe the frequency and distribution of the outcome, primary exposure, and potential covariates. Variables were investigated as potential confounders in stratified analysis to compare the crude association between HIV status and time to death with the stratified estimate. Variables were included in the multivariable model if they were significant at the alpha 0.05 level or if they changed the primary exposure beta estimate by more than 20%. Covariates known to be confounders from the literature, regardless of significance, were retained. The proportional hazards assumption was assessed by creating interaction terms for the covariates and time. Time to event analysis was performed using Kaplan-Meier and Cox proportional hazard methods. The primary endpoint was mortality after cancer diagnosis.

Statistical analyses were carried out using SAS version 9.3 (SAS Institute Inc., Cary, NC, USA). Institutional Review Boards at each site approved the protocol.

RESULTS

Cohort characteristics

There were 54 patients with a dual diagnosis of prostate cancer and HIV; 49 had full clinical data available. Total follow-up time was 159 person-years; median follow-up time was 2.7 years (interquartile range (IQR) 1.4–3.9). Patient characteristics are displayed in Table 1. The median age of the patients was 61 years (IQR, 56–67); 92% were African American. HIV risk factors included heterosexual contact (37%), injection drug use (41%), and male-to-male sexual contact (8%). The median duration of HIV infection prior to prostate cancer diagnosis was 10.5 years (IQR, 5–15). Nearly all patients (92%) were active or former

smokers. There was a high rate of hepatitis C virus co-infection (51%). At the time of cancer diagnosis, the median CD4 count was 391 cells/mm³ (IQR, 301–634), and 76% had HIV viral loads < 400 copies/ml. Most patients (82%) were taking ART at cancer diagnosis, while an additional 14% initiated ART after cancer diagnosis. Of the 40 patients taking ART at cancer diagnosis, 23 (49%) were on a PI-based regimen and 20 (43%) were on a non-nucleoside reverse transcriptase inhibitor (NNRTI)-based regimen.

Prostate cancer characteristics

Eight patients (16%) had a family history of prostate cancer. Indications for prostate biopsy included elevated PSA (76%) and abnormal screening digital rectal exam (DRE) (24%). Of the patients with an initially elevated PSA (n=37), subsequent DRE performed by a urologist revealed no focal abnormality in 10 (27%), enlargement in 19 (51%), and nodule/mass in the remaining 8 (22%). Thus, 20% of the total and 27% with an elevated PSA had a normal DRE exam.

At cancer diagnosis the mean PSA was 82 ng/ml (range, 2.5–1830), and the median was 7.7 ng/ml (IQR 5.4–18.8). The mean and median Gleason score were both 7 (range, 5–10). Thirty-four patients (70%) had clinical stage T1–2, while 15 (30%) had T3–4 disease. Anatomic disease stage was divided with 63% stage I/II and 37% stage III/IV. There were no significant differences between HIV-infected patients presenting with stage I/II and III/IV disease. A smaller proportion of patients with stage III/IV disease were taking PIs at cancer diagnosis (31%) compared to those with stage I/II disease (50%), but the difference was not statistically significant (p=0.31). The indication for prostate biopsy in the majority of patients was the same, elevated PSA, in both groups; however, more patients with stage I/II disease presented with a nodule/mass (19%), while more patients with stage III/IV disease presented with an enlarged prostate (22%), although the differences were not statistically significant (p=0.14). Patients with stage III/IV disease had higher median PSAs (16.0 vs. 7.2, p=0.01) and higher median Gleason scores (8.5 vs. 7, p<0.001) than those with stage I/II disease.

Prostate cancer treatment and outcomes

Cancer treatment regimens varied and included androgen deprivation therapy (51%), external beam radiation (51%), brachytherapy (25%), orchiectomy (4%), radical prostatectomy (22%), systemic chemotherapy (12%), and active surveillance/watchful waiting (10%). Patients with stage III–IV disease were more likely to receive systemic chemotherapy and androgen deprivation therapies (p=0.001 for each) than those with stage I–II disease.

By the end of the study period, 13 (27%) patients had died (8 (16%) from their cancer). Cumulative 5-year mortality was 22%, while cancer specific survival was 74% at 5 years. Mortality was higher in patients with stage III–IV disease compared to stage I–II disease (Figure 1A, p=0.03). Survival was not different by indication for biopsy (i.e. elevated PSA vs. abnormal DRE finding) and is shown in Figure 1B (p=0.76).

Comparison to the general population

Differences between this HIV-infected cohort and patients seen at UMGCC are shown in Table 2. HIV-infected patients presented at a younger median age (60.7 years) compared to the general population (64.0 years) ($p=0.04$) and had a significantly higher proportion of African Americans (92% vs. 45%, $p<0.001$). HIV-infected patients had significantly higher odds of presenting with more advanced stage (III/IV) disease than the general population ($p<0.001$). Overall mortality was also significantly worse in HIV-infected patients ($p=0.006$, Figure 1C).

HIV infection was associated with higher odds of advanced stage (III/IV) disease at presentation (odds ratio 2.82, 95% CI 1.52–5.21) even after controlling for race. Age, race, HIV infection, and cancer stage were all associated with increased hazard of mortality in the bivariate models. All variables, including HIV infection (adjusted hazard ratio, 2.02; 95% CI 1.14–3.58) remained associated with an increased rate of mortality in the multivariable Cox proportional hazards model. Sensitivity analyses were performed to assess how robust our findings were to violations of missing at random assumptions for subjects with unknown race and staging data from the cancer registry. Results from these analyses were not appreciably different from the complete case hazard ratios (results not shown).

COMMENT

As the incidence of NADCs continues to increase in the aging HIV-infected population, prostate cancer is likely to become a more visible problem over the next decade. This study found several differences in prostate cancer presentation between HIV-infected and HIV-uninfected men. One important difference was the substantial proportion of our cohort (37%) that presented with stage III–IV disease at the time of cancer diagnosis. In the general U.S. population, later stage disease typically makes up only approximately 15% of all prostate cancers.¹⁰ Higher stage disease at presentation is more common in African-American men, although still less than 20%. Prior authors have suggested anecdotally that prostate cancer could be more aggressive in HIV-infected individuals,^{12,13} and a recent epidemiologic analysis of U.S. cancer data found an increased odds for distant stage disease.¹⁸ This clinical cohort is consistent with the epidemiologic findings, as a significantly higher proportion of patients presented with advanced stages compared to the general population at our Center.

It remains unclear whether the more advanced staging in our cohort represents a more aggressive cancer phenotype in HIV infection, delayed presentation due to socioeconomic or other barriers to care, low rates of PSA screening in the clinics, racial differences, or other unmeasured factors. At least in this small study, correcting for race in the analysis did not account for the differences in staging. More research is necessary to examine the biological and sociological factors that may be impacting later stage prostate cancer presentation in HIV-infected men.

In our patients with prostate cancer, 5-year overall mortality was high and significantly impacted by stage at presentation. Other authors have suggested that treatment outcomes in HIV-infected patients are similar to the HIV negative general population.^{16,17} However,

these studies focused on early stage, localized cancer, and our cohort differed in the high proportion of later stage disease. Our results (Figure 1A) showed higher overall mortality among the HIV-infected patients for those with stage III/IV disease compared to those with earlier stage (I/II) disease, a finding consistent with other prostate cancer studies.^{8,10} Patients with later stage disease are more complicated to manage since chemotherapy is often used; concomitant ART with chemotherapy may be limited by drug-drug interactions, and the optimal strategy for management in HIV-infected patients has not been well-defined. Continuation of ART is generally recommended whenever possible after cancer diagnosis so as to avoid HIV-related complications of treatment interruption. Further work is needed to assess differences in treatment choice, toleration, and adverse effects in HIV-infected patients with prostate cancer compared to the general population.

PIs induce growth arrest and apoptosis of prostate cancer cells in vitro and in vivo,⁹ and one cohort study²¹ found an association between both long-term ART and PI use and decreased risk of prostate cancer, suggesting a protective effect of PIs on prostate cancer development in HIV-infected men. Patients with stage III/IV disease in our cohort were less likely to be taking PIs; however, it is difficult to determine if PIs had any impact on disease incidence or stage at presentation due to the small number of cases. Prior work has suggested that HIV-infected patients are less likely to be screened for prostate cancer than HIV-uninfected men.⁸ Although we were not able to assess the rates of routine PSA screening in our clinics overall, the majority of men (72%) in this cohort were detected through elevated PSA levels ordered through routine screening. This finding is in accord with that of other studies.^{16,22} In our study, only 28% were detected after abnormal DREs by primary care providers. Notably, 27% of patients with elevated PSA levels had normal DRE examinations when subsequently performed by a urologist, suggesting the potential importance of PSA screening for HIV-infected men. Routine PSA screening has recently been discouraged in the U.S.²³ due to a lack of demonstrated efficacy in reducing mortality at the population level. In our cohort, a substantial percentage of cancer cases would have been missed by screening DREs alone. As HIV-infected men are living longer, prostate cancer incidence may rise in this population, and so prostate cancer screening guidelines should be carefully evaluated in this context.

A strength of this analysis is the close examination of outcomes in an urban, HIV-infected population whose cancer outcomes are largely unreported. However, inferences to populations outside of the source for this study should be made with caution. Unaccounted barriers to care may have led to delayed presentation among the HIV-infected subjects and thus more advanced disease stage at presentation; however, many clinicians in our clinics have routinely screened with PSA testing which would be expected to detect cancers at earlier stages. We also compared basic data for the HIV-uninfected population with prostate cancer at our institution which was helpful in confirming the advanced stage at presentation and increased mortality, even after controlling for race and age, two factors known to be associated with mortality. As the data for the unexposed subjects came from a cancer registry, it is possible that some HIV-infected subjects are misclassified as HIV-uninfected; however this misclassification is most likely non-differential and therefore would attenuate our findings towards the null.

This study was limited by its retrospective nature. Also, as noted above, results may not be generalizable to all HIV-infected men with prostate cancer because the study population consists mainly of inner-city, African-Americans of low socioeconomic status. The total number of exposed (HIV-infected) patients limited the ability to detect differences in subgroup analyses. Additionally, causes of death were not available for all patients; since HIV-infected patients are at higher risk of death from other causes, competing risks may account for some of the differences in survival.

In conclusion we report findings from a cohort of men with prostate cancer and describe their clinical presentation and treatment outcomes by HIV-infection status. We found a significantly greater proportion of patients with advanced stage (III/IV) disease at presentation compared to the non-HIV population with prostate cancer at our institution. Additionally, our cohort had significantly worse mortality outcomes compared to the general population. Future studies are needed to ensure prostate cancer screening protocols are optimized for HIV-infected men.

Acknowledgements

DJR received support from a Paul Calabresi Clinical Oncology Training Program award (K12 CA126849).

References

1. Patel P, Hanson DL, Sullivan PS, Novak RM, Moorman AC, Tong TC, Holmberg SD, Brooks JT. Incidence of types of cancer among HIV-infected persons compared with the general population in the United States, 1992–2003. *Ann Intern Med.* 2008; 148:728–736. [PubMed: 18490686]
2. Silverberg MJ, Chao C, Leyden WA, Xu L, Tang B, Horberg MA, Klein D, Quesenberry CP Jr, Towner WJ, Abrams DI. HIV infection and the risk of cancers with and without a known infectious cause. *AIDS.* 2009; 23:2337–2345. [PubMed: 19741479]
3. Bedimo RJ, McGinnis KA, Dunlap M, Rodriguez-Barradas MC, Justice AC. Incidence of non-AIDS-defining malignancies in HIV-infected versus noninfected patients in the HAART era: impact of immunosuppression. *J Acquir Immune Defic Syndr.* 2009; 52:203–208. [PubMed: 19617846]
4. Marcus JL, Chao CR, Leyden WA, Xu L, Klein DB, Horberg MA, Towner WJ, Quesenberry CP Jr, Abrams DI, Van Den Eeden SK, et al. Prostate Cancer Incidence and Prostate-Specific Antigen Testing Among HIV-Positive and HIV-Negative Men. *J Acquir Immune Defic Syndr.* 2014; 66:495–502. [PubMed: 24820107]
5. Crum NF, Spencer CR, Amling CL. Prostate carcinoma among men with human immunodeficiency virus infection. *Cancer.* 2004; 101:294–299. [PubMed: 15241826]
6. Hessel NA, Pipkin S, Schwarcz S, Cress RD, Bacchetti P, Scheer S. The impact of highly active antiretroviral therapy on non-AIDS-defining cancers among adults with AIDS. *Am J Epidemiol.* 2007; 165:1143–1153. [PubMed: 17344204]
7. Hsiao W, Anastasia K, Hall J, Goodman M, Rimland D, Ritenour CW, Issa MM. Association between HIV status and positive prostate biopsy in a study of US veterans. *Scientific World Journal.* 2009; 9:102–108. [PubMed: 19219374]
8. Shiels MS, Goedert JJ, Moore R, Platz EA, Engels EA. Reduced risk of prostate cancer in U.S. men with AIDS. *Cancer Epidemiol Biomarkers Prev.* 2010
9. Yang Y, Ikezoe T, Takeuchi T, Adachi Y, Ohtsuki Y, Takeuchi S, Koeffler HP, Taguchi H. HIV-1 protease inhibitor induces growth arrest and apoptosis of human prostate cancer LNCaP cells in vitro and in vivo in conjunction with blockade of androgen receptor STAT3 and AKT signaling. *Cancer Sci.* 2005; 96:425–433. [PubMed: 16053514]
10. Jemal A, Bray F, Center MM, Ferlay J, Ward E, Forman D. Global cancer statistics. *CA Cancer J Clin.* 2011; 61:69–90. [PubMed: 21296855]

11. Centers for Disease Control and Prevention: HIV Surveillance Report. 2011; 23
12. Schwartz JD, Prince D. prostate cancer in HIV infection. *AIDS*. 1996; 10:797–798. [PubMed: 8805875]
13. Manfredi R, Fulgaro C, Sabbatani S, Dentale N, Legnani G. Disseminated, lethal prostate cancer during human immunodeficiency virus infection presenting with non-specific features. Open questions for urologists, oncologists, and infectious disease specialists. *Cancer Detect Prev*. 2006; 30:20–23. [PubMed: 16455210]
14. Levinson A, Nagler EA, Lowe FC. Approach to management of clinically localized prostate cancer in patients with human immunodeficiency virus. *Urology*. 2005; 65:91–94. [PubMed: 15667871]
15. O'Connor JK, Nedzi LA, Zakris EL. Prostate adenocarcinoma and human immunodeficiency virus: report of three cases and review of the literature. *Clin Genitourin Cancer*. 2006; 5:85–88. [PubMed: 16859585]
16. Pantanowitz L, Bohac G, Cooley TP, Aboulafia D, Dezube BJ. Human immunodeficiency virus-associated prostate cancer: clinicopathological findings and outcome in a multi-institutional study. *BJU Int*. 2008; 101:1519–1523. [PubMed: 18384640]
17. Kahn S, Jani A, Edelman S, Rossi P, Godette K, Landry J, Anderson C. Matched cohort analysis of outcomes of definitive radiotherapy for prostate cancer in human immunodeficiency virus-positive patients. *Int J Radiat Oncol Biol Phys*. 2012; 83:16–21. [PubMed: 22104362]
18. Shiels, MS.; Copeland, G.; Goodman, M.; Lynch, CF.; Pawlish, KS.; Pfeiffer, RM.; Snyder, JJ.; Engels, EA. Cancer stage at diagnosis in HIV-infected individuals and transplant recipients. Conference On Retroviruses And Opportunistic Infections; Boston, MA. 2014.
19. Schroder FH, Hugosson J, Roobol MJ, Tammela TL, Ciatto S, Nelen V, Kwiatkowski M, Lujan M, Lilja H, Zappa M, et al. Prostate-cancer mortality at 11 years of follow-up. *N Engl J Med*. 2012; 366:981–990. [PubMed: 22417251]
20. Riedel DJ, Mwangi EI, Fantry LE, Alexander C, Hossain MB, Pauza CD, Redfield RR, Gilliam BL. High cancer-related mortality in an urban, predominantly African-American, HIV-infected population. *AIDS*. 2013; 27:1109–1117. [PubMed: 23262503]
21. Chao C, Leyden WA, Xu L, Horberg MA, Klein D, Towner WJ, Quesenberry CP Jr, Abrams DI, Silverberg MJ. Exposure to antiretroviral therapy and risk of cancer in HIV-infected persons. *AIDS*. 2012; 26:2223–2231. [PubMed: 22951631]
22. Silberstein J, Downs T, Lakin C, Kane CJ. HIV and prostate cancer: a systematic review of the literature. *Prostate Cancer Prostatic Dis*. 2009; 12:6–12. [PubMed: 18711409]
23. Moyer VA. Force USPST: Screening for prostate cancer: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2012; 157:120–134. [PubMed: 22801674]

A

B

C

Figure 1.

A Kaplan Meier plot, stratified by stage at diagnosis (III and IV vs. I and II) ($p = 0.03$); Figure 1B Kaplan Meier plot, stratified by indication for prostate biopsy (elevated PSA vs. abnormal DRE) ($p = 0.76$); Figure 1C Kaplan Meier plot, stratified by HIV-uninfected (Group 1) vs. HIV-infected (Group 2) ($p=0.006$).

Table 1

Demographic and clinical (HIV and tumor) characteristics of prostate cancer cases.

Characteristic	Total population
No. (%)	49
Median Age [years (IQR)]	60.7 (55.8–67.2)
Race [n (%)]	
White	4 (8)
African-American	45 (92)
Family history of prostate cancer	8 (16)
HIV transmission risk factor [n (%)]	
Heterosexual	18 (37)
Injection drug use	20 (41)
MSM	4 (8)
Unknown	7 (14)
Median duration of HIV infection before cancer diagnosis [years (IQR)]	10.5 (5.0–14.5)
Smoking [n (%)]	
Current	27 (55)
Former	18 (37)
Never	4 (8)
Alcohol [n (%)]	
Current	10 (20)
Former	24 (49)
Never	15 (31)
Injection drug use [n (%)]	
Current	1 (2)
Former	19 (39)
Never	29 (59)
Hepatitis C infection [n (%)]	25 (51)
Median CD4 cell count (cells/mm ³) [IQR]	391 (301–634)
AIDS before cancer diagnosis [n (%)]	18 (37)
HIV-1 RNA viral load ^a [n (%)]	
400 copies/ml	37 (76)
> 400 copies/ml	8 (16)
Antiretroviral therapy [n (%)]	
Taking at time of cancer diagnosis	40 (82)

Characteristic	Total population
Initiated after cancer diagnosis	7 (14)
Never taking	2 (4)
ART among 40 patients at cancer diagnosis [n (%)]	
NRTI only	1 (2)
NNRTI-based	20 (43)
PI-based	23 (49)
INSTI-based	1 (2)
Salvage	2 (4)
Indication for prostate biopsy	
Elevated PSA	37 (76)
Enlarged prostate	5 (10)
Nodule or mass	7 (14)
DRE findings in 37 patients with elevated PSA	
Normal	10 (27)
Enlarged prostate	19 (51)
Nodule/mass	8 (22)
Median time to biopsy [months (IQR)]	4 (2–6)
Median PSA at cancer diagnosis [ng/ml (IQR)]	7.7 (5.4 –18.8)
Median Gleason score (IQR)	7 (6–8)
Clinical Stage [n (%)]	
T1	20 (41)
T2	14 (29)
T3	10 (20)
T4	5 (10)
Anatomic Stage [n (%)]	
I	7 (14)
II	24 (49)
III	9 (18)
IV	9 (18)
Cancer treatment [n (%)]	
Systemic chemotherapy	6 (12)
Androgen deprivation therapy ^b	25 (51)
Orchiectomy	2 (4)
External beam radiation	25 (51)
Brachytherapy	12 (25)
Radical prostatectomy	11 (22)
Watchful waiting	5 (10)

Characteristic	Total population
Outcome	
Alive	36 (73)
Dead^c	13 (27)

IQR, interquartile range; *MSM*, men who have sex with men; *ART*, antiretroviral therapy; *INSTI*, integrase strand transfer inhibitor; *NRTI*, nucleoside reverse transcriptase inhibitor; *NNRTI*, non-nucleoside reverse transcriptase inhibitor; *PI*, protease inhibitor; *PSA*, prostate specific antigen; *DRE*, digital rectal exam.

^a Missing observations: HIV-1 RNA viral load (4); hepatitis C virus (6)

^b Hormonal therapies included leuprolide acetate, bicalutamide, ketoconazole, goserelin acetate or diethylstilbestrol.

^c 8 deaths caused by prostate cancer.

Table 2

Comparison of HIV-infected patients with prostate cancer to all prostate cancer cases.

Characteristic	Total population	HIV-infected patients	<i>p</i> -value ^a
No.	1545	49	
Median Age [years (IQR)]	64.0 (58.0–70.0)	60.7 (55.8–67.2)	0.04
Race [n (%)]			
White	802 (52)	4 (8)	<0.001
African-American	698 (45)	45 (92)	
Anatomic Stage [n (%)]^b			
I	70 (5)	7 (14)	<0.001
II	1056 (68)	24 (49)	
III	111 (7)	9 (18)	
IV	108 (7)	9 (18)	
Outcome			
Alive	1203 (78)	36 (73)	0.47
Dead	342 (22)	13 (27)	

IQR, interquartile range.

^a *P*-values compare variables between total population and HIV-infected patients. Chi-square or Fisher exact test for categorical variables; Kruskal–Wallis test for continuous variables.

^b Stage information missing for 200 subjects in Total population.

Table 3
Bivariate and multivariable Cox proportional hazards models for death after diagnosis by baseline characteristics.

Characteristic	Crude HR	95% CI	p-value	Adjusted HR	95% CI	p-value
HIV Status						
Negative	1	-	-	1	-	-
Positive	2.15	1.23 – 3.75	0.02	2.02	1.14 – 3.58	0.02
Race						
White	1	-	-	1	-	-
Black	1.56	1.27 – 1.93	<0.0001	1.47	1.16 – 1.84	0.001
Tumor Stage						
I or II	1	-	-	1	-	-
III or IV	2.75	2.14	3.54	3.28	2.53 – 4.25	<0.0001
Age Group						
<58	1	-	-	1	-	-
58 – 64	1.36	0.96 – 1.91	0.08	1.37	0.95 – 1.98	0.10
65 – 70	1.52	1.09 – 2.13	0.01	1.87	1.30 – 2.70	0.0008
>70	2.69	1.96 – 3.70	<0.0001	3.4	2.40 – 4.80	<0.0001

HR, hazard ratio; CI, confidence interval.