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Incidence and risk factors for verrucae in women

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Abstract

Objectives—To describe the incidence and risk factors for verrucae in HIV-infected and uninfected women.

Design and Methods—A prospective study of 1,790 HIV-infected and 772 uninfected women. Skin examinations and interviews were performed every six months over an 8-year study period. Data collected at each visit included antiretroviral therapy use since the prior visit, CD4 counts, HIV RNA loads, and location, description, and diagnosis of verrucae. Incidence rates of cutaneous and anogenital warts were determined.

Results—Unadjusted cumulative incidence of cutaneous warts for HIV-uninfected women was 6.6%, 6.7% for HIV-infected women who initiated HAART, and 8.4% for HIV-infected, HAART-naïve women. The unadjusted cumulative incidence of anogenital verrucae for HIV-uninfected women was 9.3%, 28.4% for HIV-infected women who initiated HAART, and 25.1% for HIV-infected women who were HAART-naïve. Multivariate proportional hazard models revealed the following significant factors for the development of cutaneous verrucae among HIV-infected women: Black race (RH=0.50) and Hispanic ethnicity (RH=0.38), compared to White race. Risk factors for anogenital verrucae were: more recent recruitment (RH=0.63), HPV infection at baseline (RH=1.85), decade of age (RH=0.82), current smoker (RH=1.40), lowest CD4 count (per 100 cells/mm^3) in the past 4 years (RH=0.85), and $\log_{10}$ higher HIV viral load at the prior visit (RH=1.34).

Conclusions—HIV-infected women had a significantly increased cumulative incidence of anogenital verrucae compared to HIV-uninfected women. Although HAART did not alter the risk of developing skin or anogenital warts, those with higher CD4 cell counts and lower HIV RNA had a lower risk of developing anogenital warts.

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Keywords

HAART; HIV infection; incidence; risk factors; verrucae; women

Introduction

Patients with HIV-1 infection have an increased frequency and concomitance of dermatological diseases [1-4]. Among HIV-infected women, there is a 59% prevalence of nongenital skin disease [2,5]. Previous studies of participants in the Women's Interagency HIV Study (WIHS) reported no significant difference in the baseline prevalence of cutaneous verrucae among HIV-infected and uninfected women [2] while the prevalence of both oral and anogenital verrucae were found to be significantly higher among HIV-infected women [6-8].

Unlike Kaposi's sarcoma and eosinophilic folliculitis that improve with increased host immunity and highly active antiretroviral therapy (HAART) [9-14], cutaneous warts have been observed to be clinically recalcitrant to increasing CD4 counts and improved immunity [1, 15]. However, no cohort studies have evaluated the effects of HAART on cutaneous warts.

HAART has been shown to reduce the incidence of genital warts [16]. Although there has been conflicting data regarding treatment with HAART and HPV infections in anogenital locations, particularly regarding the persistence of HPV infection, and the regression and progression rates of cervical and anal dysplasia [17-19].

Our study objectives were to describe the incidence and risk factors, including the effect of HAART, for the development of cutaneous and anogenital verrucae in the WIHS.

Methods

The WIHS is a prospective study of HIV-1 infection in women, conducted in New York City, Washington D.C., Chicago, Southern California and the San Francisco Bay Area. The WIHS methods and baseline cohort characteristics have been previously described [20]. Briefly, between October 1994 and November 1995, 2056 HIV-1 infected and 569 uninfected women were enrolled. A second enrollment between October 2001 and September 2002, added 737 HIV-infected and 406 HIV-uninfected women. For the second wave of enrollees, medical record abstraction was performed for participants reporting HAART use at enrollment, to determine their pre-HAART CD4 and HIV RNA counts, and to verify date of HAART initiation and regimen [21]. Study protocols were reviewed and approved by the institutional review boards and informed consent was obtained from the participants.

Every six months, WIHS participants were interviewed and received a physical examination. Multiple gynecologic and blood specimens were collected at each visit. Interviewers assessed self-reported HAART use during the period prior to the study visit. Clinicians performed participants' skin examinations, including genital and oral exams. The location, description, and diagnosis of verrucae were based on clinical appearance. Beginning in October 2002, the recording of oral warts changed. Due to the change in reporting method and the overall low number of events, we did not examine predictors of incidence for oral warts.

Baseline characteristics examined included HIV status, age, race/ethnicity, marital status, parity, and lifetime number of male partners. Race/ethnicity was categorized as white, African-American, Hispanic or other ethnicity. The definition of HAART was guided by the DHHS/Kaiser Panel guidelines [22] and categorized as ever initiated HAART use or currently on HAART. The CD4+ cell count, per 100 cells/mm^3, and HIV RNA viral load, rescaled to $\log_{10}$ copies, were obtained from the visit prior if it occurred within 270 days. Exfoliated cells

for HPV DNA testing were obtained using cervicovaginal lavage (CVL) fluid [23,24]. HPV DNA was detected with L1 consensus primer polymerase chain reaction assays. Details of these laboratory methods have been published previously [25], and the results were shown to have high reproducibility, sensitivity, and specificity [24,26,27]. Additional variables examined for each study visit included: cigarette use, and incident self-reported diagnosis of clinical AIDS.

Participants who HIV-seroconverted during the study (N=16) were excluded from analysis. An incident event was defined as the first occurrence of either a cutaneous or anogenital wart after the baseline study visit. The time to event was the time from baseline until the visit date when the incident event occurred. Women with oral, skin or anogenital warts at baseline were excluded from incident events. Women who had multiple incident events in the same location were counted at the time of first occurrence only. The development of a verruca in one location did not exclude a participant from contributing to an incident event in another location.

For women in the original cohort, we defined a participant's baseline visit (time=0) as the first visit that occurred post-January 1, 1996, to correspond to HAART availability. For the 2001-02 recruits, the baseline visit occurs between October 1, 2001 and September 30, 2002. Time to event in years was calculated from time=0 to presentation of an event or last date seen (maximum date March 31, 2004) for participants who did not develop warts. Unadjusted incidence rates for skin and anogenital warts were calculated using the Nelson-Aalen estimate, stratified into one of three groups: HIV-uninfected women; HIV-infected HAART-naïve women; and HIV-infected HAART initiators.

To examine predictors of incidence, univariate and multivariate Cox proportional hazard models with time-dependent covariates were used. Multivariate models were constructed by including all covariates that were considered of clinical interest.

For analyses that assessed risk factors for skin or anogenital warts, we restricted the study sample to those women who were HIV-infected. Because women with the strongest indication for antiretroviral therapy use are more likely to use it [28], we examined the association with therapy using a version of the marginal structural proportional hazards model [29]. The weights were constructed at each visit by using pooled logistic regression models to estimate the probability of HAART initiation using both time-fixed and time-varying covariates, including a quadratic polynomial to model the effect of time. HAART initiation can be predicted much more reliably than current use; once a participant initiates HAART, she is treated as always exposed.

The time-varying predictors used to construct the weights for the marginal structural model were: visit, development of clinical AIDS, number of hospitalizations, crack, cocaine and/or heroin (CCH) use, HIV RNA at the prior visit, CD4+ cell count at the prior visit, and, in the prior 4 years, CD4 nadir and highest HIV RNA. The following time-fixed predictors were also used: phase of cohort enrollment, race, and baseline measurements of HPV infection, number of sex partners, smoking status (current/other) at baseline and age. The final, weighted model included only the time fixed covariates and a time-varying variable for initiation of HAART.

The covariates in these models were chosen on the basis of their ability to predict HAART initiation. Participants reporting use of CCH during the previous four years were indicated to have used CCH. Injection drug use (IDU) was coded in a similar way. The highest HIV RNA measurement (rescaled to $\log_{10}$) and the lowest CD4+ cell count (per 100 cells/mm^3) from the past four years were also included.

By construction the marginal structural model is focused on the estimation of the effect of HAART initiation, and not useful to examine the effects of other predictors of outcome. For

this purpose we used an additional conventional proportional hazards model, including an additional time-varying covariate for injection drug use, an additional risk factor for incidence of skin warts.

Results

A total of 1,790 (70%) HIV-infected and 772 (30%) HIV-uninfected women were evaluated for verrucae over 8 years of study follow-up. Baseline characteristics for each analytic group are provided in Table 1.

The unadjusted cumulative incidence of cutaneous warts over the 8-year study period for HIV-uninfected women was 6.6% (95% confidence interval [CI] 3.8-9.3%), 6.7% (95% CI 4.6-8.8%) for HIV-infected women who initiated HAART, and 8.4% (95% CI 4.5-12.3%) for HIV-infected women who were HAART-naive.

In both the weighted univariate model and the marginal structural model, the HAART initiator variable was highly non-significant for risk of skin warts. Results from the conventional proportional hazard model, which was run as a check, also found the HAART exposure variable to be non-significant, whether HAART was classified as current HAART use or HAART initiation.

The effects of the remaining covariates were examined in a conventional (non-weighted) proportional hazards regression model. Both black and Hispanic women had significantly less risk than whites (Table 2), and there was a suggestion that CCH use during the last four years was also a risk factor.

After 8 years of follow-up, the unadjusted cumulative incidence of anogenital verrucae for HIV-uninfected women was 9.3% (95% CI 6.3-12.2%), 28.4% (95% CI 21.7-34.5%) for HIV-infected women who initiated HAART, and 25.1% (95% CI 18.4-31.2%) for HIV-infected women who were HAART-naive.

For the marginal structural model and the conventional proportional hazard model assessing the risk of anogenital warts, HAART initiation was not significant. The HAART exposure variable was also non-significant for the univariate model and for the conventional model with current HAART use as the exposure variable.

In the conventional proportional hazards model (Table 2), HPV infection was a highly significant predictor of incident anogenital warts. There was some evidence that more than 50 sex partners was also a risk factor. Increasing age was protective, while current smoking was an additional risk factor. Finally, nadir CD4 cell count, and current viral load were highly significant risk factors.

Discussion

Verrucae were diagnosed more frequently and the 8-year unadjusted incidence rate of anogenital verrucae was higher in HIV-infected participants compared to HIV-uninfected participants.

We applied a statistical approach that only recently has been used for controlling confounders in studies of HIV/AIDS. We were able to reduce bias due to selection by indication by constructing weights (also known as propensity scores) based on prediction of HAART initiation at each visit for each individual, and including this information in a marginal structural model.

As noted in previous studies, the effect of HAART and the development of verrucae are not well understood. We found that HIV-infected women who reported initiating HAART were neither more nor less likely to develop cutaneous or anogenital verrucae when compared to those women who did not initiate HAART therapy [2,30].

Previous studies showed a reduction in the incidence of genital warts and a favorable response of genital verrucae to HAART [8,16]. We found no change in the risk of anogenital warts and the use of HAART. We could not comment on whether individual verrucae responded favorably or unfavorably to antiretroviral therapy, or detect if verrucae persisted or erupted in relation to HAART initiation.

Our findings suggest that increased CD4 cell counts and decreased HIV RNA independently reduce the risk of developing anogenital verrucae. Although HAART use was not significant for risk of anogenital warts, these changes in immunologic and virologic parameters are very likely due to use of effective HIV therapy and suggest that HAART responders (rather than all HAART users) have a decreased risk of disease.

As with incidence data based on periodic physical examinations, a detection bias exists for our outcome variables. Verrucae that may have occurred between study visits and were successfully treated or resolved on their own would have been missed.

In summary, HIV-infected women were more likely to develop anogenital verrucae than uninfected women. Although HAART did not alter the risk of developing skin or anogenital warts, those with higher CD4 cell counts and lower HIV RNA had a lower risk of developing anogenital warts. This study also confirmed the strong association with HPV infection, cigarette smoking and younger age and risk of anogenital warts.

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References

1. Berger TG, Obuch ML, Goldschmidt RH. Dermatologic manifestations of HIV infection. Am Fam Physician 1990;41:1729–1742. [PubMed: 2190455]
2. Mirmirani P, Hessol NA, Maurer TA, et al. Prevalence and predictors of skin disease in the Women's Interagency HIV Study (WIHS). J Am Acad Dermatol 2001;44:785–788. [PubMed: 11312425]
3. Dann F, Tabibian P. Cutaneous diseases in human immunodeficiency virus-infected patients referred to the UCLA Immunosuppression Skin Clinic: reasons for referral and management of select diseases. Cutis 1995;55:85–88. [PubMed: 7729163]
4. LeBoit PE. Dermatopathologic findings in patients infected with HIV. Dermatol Clin 1992;10:59–71. [PubMed: 1730173]
5. Barton JC, Buchness MR. Nongenital dermatologic disease in HIV-infected women. J Am Acad Dermatol 1999;40:938–948. [PubMed: 10365925]
6. ShiboskiCHiltonJGreenspanD, al e. HIV-related oral manifestations in two cohorts of women in San Francisco. J Acquired Immune Defi Synd19947964971
7. Silverberg MJ, Ahdieh L, Munoz A, et al. The impact of HIV infection and immunodeficiency on human papillomavirus type 6 or 11 infection and on genital warts. Sex Transm Dis 2002;29:427–435. [PubMed: 12172526]
8. Massad LS, Silverberg MJ, Springer G, et al. Effect of antiretroviral therapy on the incidence of genital warts and vulvar neoplasia among women with the human immunodeficiency virus. Am J Obstet Gynecol 2004;190:1241–1248. [PubMed: 15167825]
9. DezubeBJPantanowitzLAboulafiaDMManagement of AIDS-related Kaposi sarcoma: advances in target discovery and treatment. AIDS Read200414236238, 243-234, 251-233. [PubMed: 15199854]
10. Hermans P. Kaposi's sarcoma in HIV-infected patients: treatment options. HIV Med 2000;1:137–142. [PubMed: 11737340]
11. Mocroft A, Kirk O, Clumeck N, et al. The changing pattern of Kaposi sarcoma in patients with HIV, 1994-2003: the EuroSIDA Study. Cancer 2004;100:2644–2654. [PubMed: 15197808]
12. Costner M, Cockerell CJ. The changing spectrum of the cutaneous manifestations of HIV disease. Arch Dermatol 1998;134:1290–1292. [PubMed: 9801690]
13. Ellis E, Scheinfeld N. Eosinophilic pustular folliculitis: a comprehensive review of treatment options. Am J Clin Dermatol 2004;5:189–197. [PubMed: 15186198]
14. Highly active antiretroviral therapy and incidence of cancer in human immunodeficiency virus-infected adults. International Collaboration on HIV and Cancer. J Natl Cancer Inst 2000;92:1823–1830. [PubMed: 11078759]
15. Rodrigues LK, Baker T, Maurer T. Cutaneous warts in HIV-positive patients undergoing highly active antiretroviral therapy. Arch Dermatol 2001;137:1103–1104. [PubMed: 11493111]
16. Orlando G, Fasolo MM, Signori R, Schiavini M, Casella A, Cargnel A. Impact of highly active antiretroviral therapy on clinical evolution of genital warts in HIV-infected patients. AIDS 1999;13:291–293. [PubMed: 10202844]
17. Heard I, Schmitz V, Costagliola D, Orth G, Kazatchkine MD. Early regression of cervical lesions in HIV-seropositive women receiving highly active antiretroviral therapy. AIDS 1998;12:1459–1464. [PubMed: 9727566]
18. Lillo FB, Ferrari D, Veglia F, et al. Human papillomavirus infection and associated cervical disease in human immunodeficiency virus-infected women: effect of highly active antiretroviral therapy. J Infect Dis 2001;184:547–551. [PubMed: 11494160]
19. Ahdieh-Grant L, Li R, Levine AM, et al. Highly active antiretroviral therapy and cervical squamous intraepithelial lesions in human immunodeficiency virus-positive women. J Natl Cancer Inst 2004;96:1070–1076. [PubMed: 15265968]
20. Barkan SE, Melnick SL, Preston-Martin S, et al. The Women's Interagency HIV Study. Epidemiology 1998;9:117–125. [PubMed: 9504278]

21. Bacon MC, von Wyl V, Alden C, et al. The Women's Interagency HIV Study: an observational cohort brings clinical sciences to the bench. Clin Diagn Lab Immunol 2005;12:1013–1019. [PubMed: 16148165]

22. Department of Health and Human Services. 2004. Guidelines for the Use of Antiretroviral Agents in HIV-1-Infected Adults and Adolescents.

23. Palefsky JM, Holly EA, Ralston ML, Jay N. Prevalence and risk factors for human papillomavirus infection of the anal canal in human immunodeficiency virus (HIV)-positive and HIV-negative homosexual men. J Infect Dis 1998;177:361–367. [PubMed: 9466522]

24. Palefsky JM, Minkoff H, Kalish LA, et al. Cervicovaginal human papillomavirus infection in human immunodeficiency virus-1 (HIV)-positive and high-risk HIV-negative women. J Natl Cancer Inst 1999;91:226–236. [PubMed: 10037100]

25. Burk RD, Ho GY, Beardsley L, Lempa M, Peters M, Bierman R. Sexual behavior and partner characteristics are the predominant risk factors for genital human papillomavirus infection in young women. J Infect Dis 1996;174:679–689. [PubMed: 8843203]

26. Qu W, Jiang G, Cruz Y, et al. PCR detection of human papillomavirus: comparison between MY09/MY11 and GP5+/GP6+ primer systems. J Clin Microbiol 1997;35:1304–1310. [PubMed: 9163434]

27. Jiang G, Qu W, Ruan H, Burk RD. Elimination of false-positive signals in enhanced chemiluminescence (ECL) detection of amplified HPV DNA from clinical samples. Biotechniques 1995;19:566–568. [PubMed: 8777045]

28. Ahdieh L, Gange SJ, Greenblatt R, et al. Selection by indication of potent antiretroviral therapy use in a large cohort of women infected with human immunodeficiency virus. Am J Epidemiol 2000;152:923–933. [PubMed: 11092434]

29. Hernan MA, Brumback B, Robins JM. Marginal structural models to estimate the causal effect of zidovudine on the survival of HIV-positive men. Epidemiology 2000;11:561–570. [PubMed: 10955409]

30. FreytesDMArroyo-NovoaCMFigueroa-RamosMIRuiz-LebronRBStottsNABusquetsASkin disease in HIV-positive persons living in Puerto Rico. Adv Skin Wound Care200720149150, 152-146. [PubMed: 17473721]

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Table 1

Participant characteristics at time 0 among the 1,790 HIV-infected and 772 HIV-uninfected women in the WIHS, 1996-2004.

	Anogenital N=368 n (%)	**Cutaneous N=105 n (%)**	**Oral N=18 n (%)**	**No Warts N=2106 n (%)**	**All participants N=2562 n (%)**
HIV Status					
Seropositive	329 (18.4)	82 (4.6)	15 (0.8)	1395 (77.9)	1790 (69.9)
Seronegative	39 (5.1)	23 (3.0)	3 (0.4)	711 (92.1)	772 (30.1)
Cohort					
94/95 Recruits	308 (18.6)	98 (5.9)	13 (0.8)	1267 (76.7)	1652 (64.5)
01/02 Recruits	60 (6.6)	7 (0.8)	5 (0.6)	839 (92.2)	910(35.5)
Median (IQR) CD4 count*	286 (170, 488)	400 (219, 578)	358 (155, 607)	401 (232, 618)	382 (219, 599)
Median (IQR) HIV RNA*	4700 (300, 41000)	4000 (80, 19000)	2000 (80, 13000)	1100 (80, 19000)	1600 (80, 21000)
Median (IQR) HIV RNA*†	4700 (4000, 41000)	4000 (4000, 19000)	4000 (4000, 13000)	4000 (4000, 19000)	4000 (4000, 21000)
Median (IQR) age (yr)	38 (33.5, 42)	41 (35, 46)	42 (38, 47)	39 (32, 45)	39 (32, 45)
Baseline HPV infection	248 (21.2)	55 (4.7)	12 (1.0)	881 (75.3)	1170 (45.7)
Race/ethnicity					
Black	228 (16.2)	57 (4.1)	15 (1.1)	1129 (80.4)	1404 (54.8)
Hispanic	96 (13.5)	20 (2.8)	2 (0.3)	600 (84.0)	714 (27.9)
Other	7 (8.4)	2 (2.4)	1 (1.2)	73 (88.0)	83 (3.2)
White	37 (10.3)	26 (7.2)	0 (0.0)	304 (84.2)	361 (14.1)
Marital Status					
Widowed	29 (14.4)	13 (6.3)	1 (0.5)	166 (80.6)	206 (8.0)
Divorced/separated	80 (16.2)	30 (6.1)	5 (1.0)	393 (79.4)	495 (19.3)
Never married	135 (15.3)	33 (3.7)	6 (0.7)	720 (81.6)	882 (34.3)
Other	8 (7.4)	1 (0.9)	0 (0.0)	99 (91.7)	108 (4.2)
Married/living with partner	113 (13.4)	28 (3.3)	6 (0.7)	704 (83.4)	844 (32.9)
Smoking status					
Current	222 (17.1)	49 (3.8)	12 (0.9)	1033 (79.7)	1296 (50.6)
Former	47 (12.1)	20 (5.1)	4 (1.0)	325 (83.6)	389 (15.2)
Never	98 (11.2)	36 (4.1)	2 (0.2)	748 (85.4)	876 (34.2)
Median (IQR) lifetime male partners	12 (5,65)	10 (5,30)	33 (6, 120)	10 (5,30)	10 (5,35)
No of Children (n)					
≥3	129 (14.4)	29 (3.2)	10 (1.1)	739 (82.3)	898 (35.1)
2	77 (15.2)	25 (4.9)	1 (0.2)	414 (81.5)	508 (19.8)
1	91 (17.0)	25 (4.7)	5 (0.9)	426 (79.5)	536 (20.9)
0	71 (11.5)	26 (4.2)	2 (0.3)	527 (85.0)	620 (24.2)

* HIV-infected women only

† HIV RNA undetectable limit set to <4000 copies/mL, the lower limit of quantification. All viral loads tested below 4000 were recoded to 4000.

Table 2

Non-weighted proportional hazard models for the risk of cutaneous and anogenital verrucae among the 1,790 HIV-infected women in the WIHS.

Cutaneous verrucae	With CCH* use				Without CCH* Drug Use							
Variable	RH	Lower CI	Upper CI	P-value	RH	Lower CI	Upper CI	P-value	RH	Lower CI	Upper CI	P-value
Cohort (ref=01/02 Cohort)	2.12	0.74	6.11	0.165	2.13	0.74	6.14	0.161	2.31	0.80	6.68	0.124
Age at baseline, 10 year increase	1.25	0.91	1.72	0.169	1.26	0.92	1.72	0.143	1.27	0.94	1.72	0.126
Black (ref=white)	0.50	0.29	0.86	0.012	0.49	0.28	0.84	0.009	0.53	0.31	0.91	0.021
Hispanic	0.38	0.19	0.74	0.005	0.37	0.19	0.74	0.004	0.38	0.19	0.75	0.005
HPV infection at baseline	1.23	0.74	2.05	0.415	1.23	0.74	2.04	0.432	1.27	0.76	2.09	0.361
Lifetime number of sex partners at baseline (ref=0-4 partners)												
5-10 partners	0.93	0.47	1.86	0.846	0.94	0.47	1.86	0.857	0.89	0.45	1.76	0.738
11-50 partners	1.08	0.58	2.00	0.815	1.07	0.57	1.98	0.844	1.02	0.56	1.88	0.941
51+ partners	0.96	0.46	2.02	0.912	0.95	0.45	2.02	0.902	0.94	0.45	1.99	0.878
Current smoker	0.67	0.38	1.17	0.158	0.68	0.39	1.19	0.178	0.78	0.48	1.28	0.326
1 Hospitalization, prior visit (ref=0)	0.70	0.32	1.56	0.382	0.70	0.32	1.57	0.390	0.69	0.31	1.52	0.356
2+ Hospitalization, prior visit	0.63	0.15	2.62	0.527	0.63	0.15	2.64	0.529	0.62	0.15	2.54	0.501
Developed AIDS by visit	0.95	0.59	1.55	0.841	0.97	0.60	1.56	0.888	1.03	0.64	1.66	0.916
Initiated HAART	1.14	0.65	1.99	0.650	1.13	0.65	1.97	0.676	1.18	0.67	2.07	0.577
Lowest CD4, past 4 years (per 100 cells)	1.18	0.92	1.50	0.192	1.18	0.92	1.51	0.186	1.19	0.94	1.52	0.150
CD4 count, prior visit (per 100 cells)	0.87	0.73	1.04	0.127	0.87	0.73	1.04	0.126	0.87	0.73	1.03	0.113
Highest Log10 VL, past 4 years	0.98	0.72	1.34	0.896	0.98	0.72	1.35	0.907	0.97	0.71	1.32	0.829
Log10 HIV RNA, prior visit	1.09	0.83	1.43	0.538	1.09	0.83	1.43	0.547	1.10	0.84	1.44	0.490
Use of CCH*, past 4 years	1.84	0.93	3.62	0.078	1.93	1.01	3.66	0.045				
Use of CCH*, prior visit	0.38	0.12	1.23	0.106	0.50	0.20	1.22	0.129				
IDU, past 4 years	1.18	0.47	2.98	0.724								
IDU, prior visit	1.85	0.40	8.60	0.431								

Anogenital verrucae	With CCH* use				Without CCH* Drug Use							
Variable	RH	Lower CI	Upper CI	P-value	RH	Lower CI	Upper CI	P-value	RH	Lower CI	Upper CI	P-value
Cohort (ref=01/02 Cohort)	0.63	0.44	0.89	0.009	0.63	0.44	0.89	0.008	0.62	0.44	0.88	0.007
Age at baseline, 10 year increase	0.82	0.71	0.96	0.010	0.82	0.71	0.95	0.009	0.83	0.71	0.96	0.011
Black (ref=white)	1.27	0.89	1.80	0.182	1.28	0.90	1.81	0.169	1.27	0.90	1.80	0.171
Hispanic	1.15	0.78	1.69	0.482	1.17	0.80	1.72	0.425	1.17	0.80	1.72	0.423
HPV+ at baseline	1.85	1.42	2.40	<.001	1.85	1.42	2.40	<.001	1.83	1.41	2.37	<.001
Lifetime number of sex partners at baseline (ref=0-4 partners)												
5-10 partners	0.94	0.68	1.32	0.736	0.95	0.68	1.32	0.738	0.95	0.68	1.32	0.759
11-50 partners	0.86	0.63	1.19	0.370	0.87	0.63	1.19	0.380	0.88	0.64	1.21	0.431
51+ partners	1.34	0.97	1.84	0.077	1.36	0.99	1.87	0.059	1.41	1.03	1.93	0.034
Current smoker	1.40	1.08	1.81	0.012	1.39	1.08	1.81	0.012	1.40	1.10	1.80	0.008
1 Hospitalization, prior visit (ref=0)	1.33	0.99	1.79	0.055	1.32	0.99	1.78	0.061	1.33	0.99	1.79	0.058
2+ Hospitalization, prior visit	0.97	0.56	1.71	0.928	0.96	0.54	1.68	0.874	0.95	0.54	1.67	0.868
Developed AIDS by visit	1.26	0.98	1.61	0.072	1.26	0.98	1.62	0.070	1.26	0.98	1.62	0.069
Initiated HAART	1.11	0.85	1.44	0.445	1.10	0.85	1.43	0.473	1.08	0.83	1.40	0.561

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Anogenital verrucae Variable	With CCH* use				Without CCH* Drug Use							
	RH	Lower CI	Upper CI	P-value	RH	Lower CI	Upper CI	P-value	RH	Lower CI	Upper CI	P-value
Lowest CD4, past 4 years (per 100 cells)	0.85	0.77	0.95	0.002	0.85	0.77	0.94	0.002	0.85	0.77	0.95	0.003
CD4 count, prior visit (per 100 cells)	1.01	0.94	1.08	0.865	1.01	0.94	1.08	0.854	1.00	0.94	1.08	0.917
Highest Log10 VL, past 4 years	0.95	0.80	1.13	0.539	0.95	0.80	1.13	0.587	0.95	0.80	1.13	0.587
Log10 HIV RNA, prior visit	1.34	1.16	1.56	<.001	1.34	1.16	1.55	<.001	1.34	1.15	1.54	<.001
Use of CCH*, past 4 years	0.98	0.68	1.41	0.904	0.96	0.69	1.35	0.832				
Use of CCH*, prior visit	1.19	0.76	1.85	0.444	1.16	0.77	1.73	0.485				
IDU, past 4 years	0.97	0.56	1.66	0.907								
IDU, prior visit	0.83	0.37	1.86	0.657								

*CCH = crack, cocaine, or heroin use