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Trichomonas vaginalis in HIV-Infected women: A Risk Factor for High Risk Human Papillomavirus.

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Abstract

Objective: Women with *Trichomonas vaginalis* (TV) are four times as likely to have high-risk human papilloma virus (HRHPV). TV is more common in HIV-infected women and associated with HIV transmission. This study aimed to describe the association between TV and HRHPV in HIV-infected women undergoing cervical cancer screening.

Methods: We reviewed the records of 329 HIV-infected women presenting for cervical cancer screening. Chi-squared analysis was used to compare proportions of disease in women with and without HRHPV. Univariate and multivariate logistic regression analyses were used to determine factors associated with HRHPV.

Results: Of women screened for HRHPV, over half were infected (114/210). TV infection was present in 57/257 screened. TV was associated with a 4-fold increase risk of HRHPV (OR, 3.8; 95% CI 1.6-9). Current abnormal pap (OR, 15.9; 95% CI 8.1-31.3) and AIDS (OR, 6.1: 95% CI 2.6-14.5) were the most significant risk factors for HRHPV infection.

Conclusions: TV and HRHPV are common infections among HIV-infected women. TV was a significant risk factor for HRHPV infection. The consequence of untreated TV infection may be persistent HRHPV infection and risk of cervical cancer.

Keywords: *Trichomonas vaginalis*; HIV; High-risk Human Papillomavirus.

Introduction

Trichomonas vaginalis is a parasitic protozoan that causes vaginitis in women and is the most common treatable sexually transmitted infection (STI) worldwide [1,2]. The true prevalence of *Trichomonas* is likely underestimated due to a lack of reporting, low rates of screening in asymptomatic patients, and the poor sensitivity of microscopy for diagnosis [3,4]. *Trichomonas* infection affects 3.2% of American women, but non-Hispanic, black women have disproportionately high rates of *Trichomonas* compared to non-Hispanic, white women (13% vs.1%) [5,6].

Trichomonas infection is linked to significant morbidity in women. Specifically, *Trichomonas* has been associated with increased rates of preterm delivery, low birth weight in neonates, pelvic inflammatory disease (PID), cervical dysplasia, and human immunodeficiency virus (HIV) acquisition and transmission [7-11]. Assuming an association with HIV transmission, one modeling study estimated that *Trichomonas* vaginitis in women is associated with 6% of new HIV infections among susceptible partners each year in the United States [12].

The prevalence of *Trichomonas* infection among HIV-infected women is significantly higher than the general population and ranges from 17-63%. [9,13-15]. The highest incidence of *Trichomonas* infection among HIV-infected women was noted among inner city minority women using drugs [15].

HIV-infected women also have high rates of human papillomavirus (HPV) co-infection [16-18]. HIV-infected women are more likely to have persistent high risk HPV (HR HPV) infections, possibly increasing their risk of developing cervical cancer compared with immunocompetent women [19]. *Trichomonas* vaginitis has been linked to HR HPV infection in HIV uninfected women [9,20,21]. We aimed to determine an association between *Trichomonas* vaginitis and HR HPV in HIV-infected women. We hypothesized that *Trichomonas* vaginitis in HIV-infected women would increase their risk of a HR HPV infection at the time of routine cervical cancer screening.

Methods

Size and power calculation Sample

Our retrospective case-control study was approved by the Medical University of South Carolina Internal Review Board (Protocol #13184). In order to calculate sample size; we assumed that *Trichomonas* infection increases the risk of HR HPV infection in HIV-infected women by a factor of 4. This is twice the associated rate reported in HIV uninfected women. Assuming a probability of 0.8, 76 subjects (38 cases and 38 controls) were needed to reject the null hypothesis that *Trichomonas* does not increase the risk of HR HPV infection. The Type I error probability associated with this power calculation was 0.05. Assuming 25% of HIV-infected women have a *Trichomonas* infection, 304 patient charts would need to be reviewed in order to identify 76 subjects infected with *Trichomonas*.

Data collection

We reviewed the charts of 329 HIV-infected women who presented for gynecologic and cervical cancer screening between 2006 and 2013. The results of the most recent cervical cancer screening exam for each woman were collected. Cervical cancer screening was performed with liquid based cytology using ThinPrep® Imaging System (Hologic Inc., MA). Additional patient variables collected were: age, race, parity, history of abnormal cervical cytology, prior treatment for cervical dysplasia, hysterectomy, hepatitis B (HBV) and C (HCV) status, and history of other STIs. HBV status was considered positive if the patient was currently or had ever been hepatitis B antigen positive and/or was core antibody positive. Previous treatment for abnormal cervical cytology could include: cryotherapy, cold knife cone, loop excision electrode procedure, and/or hysterectomy. Cervical dysplasia treatment history was obtained from documentation of patient self-report, operative notes, and surgical pathology results.

Each subject's record was reviewed for evidence of or report of STIs prior to the most recent cervical cancer screening exam. STIs recorded as part of this study were: herpes simplex virus (HSV), *Chlamydia trachomatis (CT), Neisseria gonorrhoeae (GC),* external genital warts (EGW), *Trichomonas vaginalis*, and syphilis. Patient history of STIs was determined by documented patient self-report, previous positive nucleic acid amplification tests (NAATs), serologies, cultures, and/or microscopy examination.

Patient characteristics and results of screening tests at the time of the most recent cervical cancer screening exam were recorded. These included: current pregnancy, cervical cytology and histology, HR HPV infection determined by PCR (Cervista HPV high risk assay, Hologic, Inc.) or histology demonstrating cervical intraepithelial neoplasia, bacterial vaginosis (BV) screening, and *Trichomonas* screening. Any new STI diagnosis at the time of cervical cancer screening was noted.

BV screening was performed using Amsel's criteria or gram stain (Nugent's score ≥7) [22]. A diagnosis of BV by Amsel's criteria consists of 3 of the following 4 findings: vaginal discharge, pH greater than 4.5, positive whiff test, and presence of clue cells on microscopy [23]. *Trichomonas* screening was considered positive if organisms were identified by Gen-Probe APTIMA *Trichomonas vaginalis* assay (San Diego, CA), microscopy, culture (InPouch™ TV test, BioMed Diagnostics San Jose, CA), or cervical cytology (ThinPrep® Imaging System,Hologic Inc., MA).

To assess HIV status at time of the cervical cancer screening exam, a subject's HIV RNA viral load (copies/ml) and CD4 cell counts (cells/mm^3) within the last year were noted. RNA viral load was recorded as zero copies/ml if the HIV RNA viral load was undetectable using Abbott M2000 Real time PCR test (Abbott Park, Illinois). For subjects on anti-retroviral therapy (ARVs) and/or opportunistic infection prophylaxis (OIP), therapeutic regimen at the time of the most recent cervical cancer screening exam was recorded.

Data Analysis

Statistical analyses were performed using SAS version 9.3 (SAS® Software Cary, NC). The mean was calculated for normally distributed continuous variables (age) and the median was calculated for non-normally distributed continuous variables (viral load, parity, and CD4 cell count). Chi-square tests were used to compare the proportion of subjects affected by the outcomes of interest. Odds ratios were determined using univariate and multivariate logistic regression analyses. Variables with a p-value ≤ 0.2 in univariate analysis were included in the multivariate models.

Results

From 2006 to 2013, 329 women presented for cervical cancer screening. The mean age of these women was 44 years (± 11) and the median parity was 2 (IQR 0-9). Of 329 women, 264 were non-Hispanic, black, 18 were currently pregnant, and 46 had undergone a hysterectomy. More than half of the women had previously had abnormal cervical cytology (188/329). Thirty-six percent (67/188) of the women with abnormal cytology had been treated for cervical intraepithelial neoplasia.

Many of the women presenting for cervical cancer screening had a prior STI (211/329), HSV (79/211) and Trichomonas (108/211) were the most common historical STIs. At the most recent screening exam, abnormal cervical cytology was present in one third of women (113/329). Almost half of these women underwent cervical biopsies (54/113). Additional patient characteristics recorded are shown in Table 1.

Eighty-eight percent of the women (291/329) presenting for cervical cancer screening were screened for BV. Of the women screened, one third of women had BV (86/291). The most common STIs present at the time of the cervical cancer screening were *Trichomonas* (57/329), HSV (5/329) and *Chlamydia* (5/329).

The median viral load for women in the study was undetectable (IQR 0-1,410,696 copies/mL) and the median CD4 count was 496 cells/mm^3 (IQR 7-1746). Less than 20% of subjects (55/329) had AIDS, defined as a CD4 cell count ≤ 200 cells/mm^3.

Table 1. Characteristics of HIV-infected women undergoing cervical cancer screening.

	N=329 (%)
Age	44 (±11)
Parity	2 (0-9)
Black, non-Hispanic	264 (80%)
White, non-Hispanic	49 (15%)
White, Hispanic	15 (5%)
Pregnant	18 (5%)
History of Hysterectomy	46 (14%)
History of Abnormal Pap	188 (57%)
Prior Treatment for Abnormal [a]Pap	67 (20%)
Current Abnormal Pap	113 (34%)
Trichomonas infection	57/257 (22%)
HR HPV infection	114/210 (54%)
CD4 cell count (cells/mm^3)	496 (7-1746)
AIDS[b]	55 (17%)
HIV RNA Viral Load	0 (0-1,410,696)
Undetectable Viral Load	214 (65%)
Current use of ARVs	283 (86%)

[a]Prior treatment for abnormal cervical cytology could include: cryotherapy, cold knife cone, loop excision electrode procedure, and/or hysterectomy. [b]AIDS defined as CD4 cell count of ≤ 200 cells/mm^3.

The majority of women reported taking ARVs (283/329) and few required OIP (62/329). The proportion of women taking OIP reflected the number of women with CD4 counts ≤ 200 cells/mm^3.

Seventy-four percent of women presenting for cervical cancer screening were also screened for *Trichomonas* (237/329). Of the women screened for *Trichomonas* using recommended screening techniques (NAATs, culture or microscopy), 15% (37/237) had an infection. Notably, 20 women were incidentally found to have *Trichomonas* on cytology. When accounting for incidental *Trichomonas* infection noted on cytology, a total of 57 subjects in the study had *Trichomonas* infection.

Over half of the women who were evaluated for HR HPV infection by PCR or cervical biopsies were infected (114/210). One hundred and sixty-eight women were evaluated for both HR HPV and *Trichomonas* infection at their most recent exam. Of these women, 36 were infected with *Trichomonas*. The majority of these women were also infected with HR HPV (28/36). Because fewer women than anticipated were screened for *Trichomonas* and evaluated for HR HPV during the same exam, we identified approximately half of the desired number of subjects (36/76) with *Trichomonas* infection.

The results of chi-square analyses comparing patient variables in HIV-infected women with and without HR HPV infection are listed in Table 2. HR HPV infection was strongly related to current abnormal cervical cytology (p=<0.0001) and CD4 count ≤ 200 cells/mm^3 (p=<0.0001). *Trichomonas* (p=0.001), history of abnormal cytology (p=0.003), black race (p=0.02) and HBV infection (p=0.01) were significantly associated with HR HPV. Age, parity, hysterectomy, BV and other current STIs were not significantly related. A history of other STIs prior to the exam was also not associated with current HR HPV infection. Subjects with a history of *Trichomonas* (108/329) were more likely to

have a history of abnormal cervical cytology (p=0.02). One hundred and eighty-eight women in the study had a history of abnormal Pap smear and of those 38% (71/108) also had a history of *Trichomonas* infection.

Table 2. Comparison between HIV-infected women with and without evidence of high risk human papillomavirus during cervical cancer screening.

	HR HPV+ (n=114)	HR HPV- (n=96)	p-value
Age	44 (±10)	45 (±11)	0.2
Parity	3 (0-7)	2 (0-8)	0.1
Black, non-Hispanic	98 (86%)	70 (73%)	0.02
History of hysterectomy	11 (10%)	11 (11%)	0.7
CD4 cell count (cells/mm^3)	320 (7-1348)	579 (67-1382)	<0.0001
AIDS	37 (32%)	7 (7%)	<0.0001
Current ARV use	94 (82%)	89 (93%)	0.03
Viral Load	20 (0-1,410,696)	0 (0-51,782)	<0.0001
Undetectable viral load	57 (50%)	78 (81%)	<0.0001
Abnormal Pap	92 (81%)	20 (21%)	<0.0001
History of abnormal pap	79 (69%)	50 (52%)	0.01
Trichomonas infection (n=168)	28/91 (31%)	8/77 (10%)	0.001
BV (n=186)	33 (34%)	23 (26%)	0.3
Current STI			
HBV	26 (23%)	9 (9%)	0.01
HCV	17 (15%)	12 (13%)	0.6
HSV	2 (2%)	0	0.5
EGW	2 (2%)	0	0.5
Chlamydia	3 (3%)	0	0.3
Syphilis	1 (0.9%)	0	1.0

Means were compared using Student's t-test and reported with standard deviation. Medians were compared using Mann-U Whitney test and reported with interquartile ranges. Proportions are compared using chi-square analysis and Fischer's exact tests were applicable. There were no cases of gonorrhea at the time of cervical cancer screening.

In a univariate logistic regression analysis, current abnormal cervical cytology was the most significant risk factor for a HR HPV infection (OR 15.9, 95% CI 8.1-31.3).

Other factors associated with HR HPV infection in univariate logistic regression models were: CD4 cell ≤ 200/mm^3 (OR 6.1, 95% CI 2.6-14.5), *Trichomonas* infection (OR 3.8, 95% CI 1.6-9), black race (OR 2.3,95% CI 1.1-4.6), history of abnormal cervical cytology (OR 2.1, 95% CI 1.2-3.7) and HBV infection (OR 2.9, 95% CI 1.3-6.4). Well-controlled HIV infection, determined by an undetectable viral load, and current use of ARVs decreased the risk of HR HPV by 80% and 60% respectively (OR 0.2, 95% CI 0.1-0.4; OR 0.4, 95% CI 0.2-0.9)(See Table 3).

In a multivariate logistic regression analysis controlling for race, history of abnormal cervical cytology, current abnormal cervical cytology, *Trichomonas* infection, viral load, and CD4 ≤ 200/mm^3, the association between TV and HR HPV was attenuated and no longer significant (OR 2.1; 95% CI 0.7-6.5). In multivariate analysis, race, a history of abnormal cervical cytology and CD4 count ≤ 200 cells/mm^3 were also no longer significantly associated with HR HPV.

Current abnormal cervical cytology (OR 14.9; 95% CI 6.9-32.2) and HBV infection (OR 6.2; 95% CI 2-18.9) remained significantly associated with HR HPV in multivariate analysis. An undetectable viral load remained associated with a lower risk of HR HPV infection (OR 0.3; 95% CI 0.1-0.8) but ARV use was no longer significantly associated low risk of HR HPV (See Table 3).

Discussion

Trichomonas vaginalis infection was associated with a 4-fold increased risk of HR HPV infection in our population of HIV-infected women undergoing cervical cancer screening. Twenty-two percent of our subjects had a *Trichomonas* infection, which is consistent with prevalence data from other studies [13]. Non-Hispanic, black women and those with a CD4 cell count ≤ 200 cells/mm^3 were more likely to be infected with HR HPV compared with other HIV-infected women. When controlling for multiple factors, the association between *Trichomonas* infection and HR HPV was attenuated. This finding is most likely due to a small sample size of subjects screened for both *Trichomonas* and HR HPV during the same exam.

Based on prevalence studies in HIV-infected women, we assumed that 50% of our study population would have a HR HPV infection and 25% would be infected with *Trichomonas*. These assumptions were consistent with the rates of infection in our population (57% and 22% respectively). We had anticipated that among 304 women, we would identify 76 women infected with *Trichomonas* infection who were evaluated for HR HPV infection at a routine cervical cancer screening. However, only 36 eligible women were identified out of 329.

Table 3. Factors associated with high risk human papillomavirus among HIV-infected women.

HR HPV+	**Unadjusted**			**Adjusted**		
	OR	95% CI	p-value	OR	95% CI	p-value
Abnormal Pap	15.9	8.1-31.3	<0.0001	14.9	6.9-32.2	<0.0001
AIDS	6.1	2.6-14.5	<0.0001	3.5	1-11.9	0.04
Trichomonas infection	3.8	1.6-9	0.002	2.1	0.7-6.5	0.2
Hepatitis B Virus infection	2.9	1.3-6.4	0.01	6.2	2-18.9	0.001
Black, non-Hispanic race	2.3	1.1-4.6	0.02	1.6	0.6-3.9	0.3
History of Abnormal Pap	2.1	1.2-3.7	0.01	1.3	0.6-2.9	0.5
Current ARV use	0.4	0.2-0.9	0.03	1.7	0.4-6.9	0.5
Undetectable viral load	0.2	0.1-0.4	<0.0001	0.3	0.1-0.8	0.02

Adjusted OR were determined using multivariate analysis of variables with p-value ≤ 0.2 in univariate analysis. Adjusted OR for Trichomonas accounts for patients who were screened.

Presumably, the unexpected low number of subjects screened for both *Trichomonas* and HR HPV infections is due to the study period. Many of the subjects in this study underwent cervical cancer screening prior to 2010. The release of the CDC Sexually Transmitted Diseases Treatment Guidelines, which recommend annual *Trichomonas* screening among HIV-infected women, occurred in 2010 [24]. Previous guidelines had not recommended routine *Trichomonas* screening in any population. The 2010 guidelines made the recommendation for annual screening secondary to the high prevalence of *Trichomonas* among HIV-infected women [24]. When our study subjects are separated into women screened for HR HPV before and after January 2011, the rate of concurrent *Trichomonas* testing doubles from 43% (61/143) to 80% (149/186).

Routine HR HPV screening in HIV-infected women 30 years or older with normal cervical cytology is not yet recommended in HIV-infected women. However, there is promising evidence that HIV-infected women with elevated CD4 counts may be candidates for HPV triage screening due to similar rates of disease progression compared to HIV uninfected women [25].

Despite inadequate sample size to support our hypothesis, these data give plausibility to an association between *Trichomonas* vaginitis and HR HPV infection in HIV-infected women. There are several possible mechanisms by which *Trichomonas* increases the risk of HIV infection. These theories may be applicable to the interaction between *Trichomonas* and HR HPV infection. Immunosuppression and inflammation associated with HIV may also potentiate the role that *Trichomonas* plays in persistent HR HPV infection.

A mechanism by which *Trichomonas* may increase entry of opportunistic viruses into susceptible host cells is to weaken the mechanical barrier of the vaginal epithelium. A cysteine protease released by *Trichomonas* inactivates human secretory leukocyte protease inhibitor (SLPI) at the site of infection and triggers cell apoptosis [26,27]. Human SLPI is responsible for containing other proteases recruited during an inflammatory response. Without repression by SLPI, inflammatory proteases can degrade surrounding tissue allowing for penetrance of pathologic organisms such as HIV and HR HPV [26,28].

Trichomonas vaginitis leads to significant inflammation of the vaginal epithelium. In the face of increased inflammation from *Trichomonas*, CD4 lymphocytes are recruited to the site of infection. These cells are susceptible to HIV infection [29]. *Trichomonas* can engulf HIV-infected lymphocytes and potentially carry HIV past the protective vaginal epithelium [28,30].

In HIV-infected women, genital shedding of the HIV virus is increased in the presence of *Trichomonas* vaginitis [31]. *Trichomonas* stimulates IL-8, which activates HIV-replication through TNFα [28]. The up-regulation of HIV viral replication and genital shedding could lead to increased HIV transmission to susceptible partners. Given these plausible associations of *Trichomonas* in the acquisition and transmission of HIV, it is likely that *Trichomonas* infection increases the risk of other viral STIs such as HR HPV in HIV-infected women.

HIV immunosuppression combined with inflammation and vaginal epithelial breakdown caused by *Trichomonas* infection may create an advantageous environment for persistent HR HPV infection. If this relationship exists, routine screening and treatment of *Trichomonas* in HIV-infected patients, as recommended by CDC, may decrease the risk of persistent HR HPV and ultimately cervical cancer. The goal of diagnosis and effective treatment of *Trichomonas* vaginitis in HIV-infected women would not only be to reduce potential transmission of HIV but also to decrease the risk of persistent HR HPV infection [24]. By reducing factors that lead to persistent HR HPV infection, HIV-infected women would be less likely to develop squamous cell carcinoma of the cervix.

Future studies should investigate the role of effective *Trichomonas* treatment and control of HIV disease on persistent HR HPV infection.

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