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Prevalence and risk factors of cervical squamous intraepithelial lesions among HIV-infected women in Dar es Salaam, Tanzania

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Abstract

To determine the prevalence and predictors of cervical squamous intraepithelial lesions (SIL) among HIV-infected women in Tanzania, a cross-sectional study was conducted among HIV-infected women at HIV care and treatment clinics. A Papanicolaou (Pap) smear was used as a screening tool for detection of cervical SIL. From December 2006 to August 2009, 1365 HIV-infected women received cervical screening. The median age was 35 (interquartile range [IQR]: 30–42) years, and the median CD4 + cell count was 164 (IQR: 80–257) cells/mm^3. The prevalence of cervical SIL was 8.7% (119/1365). In multivariate analysis, older age (≥50 versus 30–<40 years: prevalence ratio [PR], 2.36; 95% confidence interval [CI], 1.45–3.84, p for trend = 0.001), lower CD4 + cell counts (<100 versus ≥200 cells/mm^3: PR, 1.55; 95% CI, 1.01–2.36, p for trend = 0.03) and cervical inflammation (PR, 1.73; 95% CI, 1.16–2.60, p = 0.008) were associated with an increased risk of cervical SIL. Women with advanced WHO HIV disease stage (IV versus I/II: PR, 3.45; 95% CI, 1.35–8.85, p for trend = 0.01) had an increased risk for high-grade SIL. In resource-limited settings where it is not feasible to provide cervical cancer prevention services to all HIV-infected women, greater efforts should focus on scaling-up services among those who are older than 50 years, with lower CD4 cell counts and advanced HIV disease stage.

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Declaration of conflicting interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Keywords

HIV; AIDS; human papillomavirus; HPV; prevalence; risk factors; cervical squamous intraepithelial lesions; Papanicolaou (Pap) smear; HIV-infected women; Tanzania

Introduction

Infection with human papillomavirus (HPV) is a well-established cause of cervical cancer.[1] HIV-infected women are more likely to have persistent, oncogenic HPV infections that lead to precancerous squamous intraepithelial lesions (SIL).[2] If left untreated, SIL may progress to cervical cancer.

Over the past decade, many countries across sub-Saharan Africa have successfully established dedicated HIV programmes to deliver antiretroviral therapy (ART). The scaling-up of global access to ART has significantly extended the lifespan of HIV-infected women in this region. With increased lifespan, HIV-infected women are at higher risk of developing cervical cancer. In order to control the high incidence of cervical cancer among HIV-infected women, substantial efforts have been made to integrate cervical cancer screening and treatment service into HIV programmes in sub-Saharan Africa.[3–6]

Tanzanian women bear the highest burden of cervical cancer in East Africa, with age-standardised incidence and mortality rates of 54.0 and 32.4 cases per 100,000 person-years.[7] However, the coverage of cervical cancer screening is quite low, and the majority of these HIV-infected women have not been screened for cervical cancer.[8,9] In this study, we aimed to determine the prevalence and risk factors of cervical SIL among HIV-infected women.

Methods

Study participants

This cross-sectional study was conducted among HIV-infected women attending HIV care and treatment clinics (CTCs) supported by the Management and Development for Health (MDH) non-governmental organisation and the United States President's Emergency Plan for AIDS Relief (PEPFAR) in Dar es Salaam, Tanzania. From December 2006 to August 2009, Papanicolaou (Pap) smears were intermittently available in a small number of the HIV CTCs. Due to limited resources and the availability of medical staff, screening rooms and equipment, the cervical cancer prevention service was only provided on certain weeks or dates across the three-year period in a small number of clinics. Women with a clinic appointment in that specific week or dates were evaluated for their eligibility for cervical cancer screening. The eligibility criteria included: (1) serologically-confirmed HIV infection, (2) age greater than 15 years old, (3) currently sexually active or sexually active in the past and (4) without a prior hysterectomy. A total of 1365 HIV-infected women patients received Pap smear screening during the study period. The Institutional Review Board at Harvard University and the National Institute for Medical Research in Tanzania approved this study.

Study procedure and data collection

All study participants received a Pap smear performed by physicians during their clinic visits. Samples were sent to the Department of Histopathology at the Muhimbili National Hospital, Dar es Salaam, for evaluation by two pathologists. The presence or absence of cervical inflammation was determined through cervical cytological examination, and SIL was determined by cervical cytology according to the Bethesda system.[10] If the results of the two pathologists reviewing the samples were discordant, a third pathologist was asked to evaluate the sample, and the three jointly came up with a report for the specimen through a panel discussion. A Pap smear result was defined as abnormal if pathologist found low-grade SIL (LSIL), high-grade SIL (HSIL) or atypical squamous cells of undetermined significance (ASCUS).

Physicians and nurses completed standard forms capturing demographic and clinical information. Anthropometric measures including height and weight were obtained by nurses using standardised techniques. Body mass index (BMI) was calculated as the patient's weight in kilograms divided by the square of the patient's height in metres. Haemoglobin levels and CD4 + cell counts were monitored four-monthly. When CD4 and haemoglobin were not measured on the same date as the date of Pap smear sampling, the most recent measures prior to Pap smear screening were used. The median time between the last available CD4 + measurement and Pap smear screening was 35 days (interquartile range [IQR]: 28–50 days).

Study variables

The primary outcomes of interest were abnormal Pap smear results, LSIL and HSIL from cytology. We considered the following variables as potential predictors for abnormal Pap smear results: age (<30, 30 to <40, 40 to <50, ≥50 years), married (no/yes), BMI (<17, 17 to <18.5, 18.5 to <25, 25 to <30, ≥30 kg/m^2), haemoglobin (<8.5, 8.5 to <10, 10–<12,≥12 g/dL), WHO HIV disease stage (I/II, III, IV), CD4+cell counts (<100, 100–<200, ≥200 cells/mm^3), calendar year of screening (2006/2007, 2008, 2009), ART (no/yes) and cervical inflammation (no/yes). The cut points for model covariates were determined based on the distribution of data to ensure that each category had enough data or we used well-established cut points when these existed, as for severe anaemia or anaemia.

Statistical analysis

Generalised estimating equations (GEE) for a binary outcome with the log link function and exchangeable working correlation structure were used to identify risk factors for abnormal Pap smear results. Using these log-binomial models, we directly estimated the prevalence ratio (PR) to quantify the effect of each possible risk factor in univariable and multivariate models.[11] Because the prevalence odds ratio is not a good estimate of the prevalence ratio when the prevalence rate is not very low, and because the prevalence of abnormal Pap smears was not rare in this study population, logistic regression was not used for estimation and inference.[12] Potential risk factors with a p value ≤0.2 in univariate analysis were included in multivariate analysis.[13] Marginal structural models were used to adjust for possible selection bias due to differences in the screened and unscreened populations

that may have affected the estimates of the effects of the risk factors on the prevalence of abnormal Pap smears.[14]

Statistical analysis was performed using statistical software package SAS, Release 9.3 (Cary, North Carolina, USA). The significance tests were two-sided, and differences were considered significant at $p < 0.05$.

Results

The median age of the women participants was 35 years (IQR: 30–42 years), and the median CD4+cell count was 164 cells/mm3 (IQR: 80–257 cells/mm^3). About one-third of the women were married, 91% were on ART and 74% were within two months after ART initiation at the time of their Pap smear screening. On cytological examination, 845 (62%) of the 1365 women were found to have cervical inflammation, and 119 (8.7%) had a cervical abnormality. Among the 119 women with abnormal Pap smear results, 53 (3.9% of 1365) had LSIL and 47 HSIL (3.4%), while the other 19 (1.4%) had ASCUS (Table 1).

After adjusting for potential confounders, women aged ≥50 years had a 2.36 increased risk of abnormal Pap smear results compared to those aged 30–<40 years (PR, 2.36; 95% confidence interval [CI], 1.45–3.84; *p* for trend=0.001). Compared to women with CD4+cell counts ≥200 cells/mm^3, those with CD4+cell counts <100 cells/mm^3 had a 55% increased risk of abnormal Pap smear results (PR, 1.55; 95% CI, 1.01–2.36; *p* for trend=0.03). Cervical inflammation was associated with higher risk of abnormal Pap smear results (PR, 1.73; 95% CI, 1.16–2.60; *p*=0.008), and women screened in 2009 had a 38% reduced risk for abnormal Pap smear results (PR, 0.62; 95% CI, 0.40–0.94; *p* for trend=0.02) compared to women screened in 2006 or 2007 (Table 2).

We examined potential risk factors for LSIL and HSIL separately. After adjusting for potential confounders, older age, cervical inflammation and calendar year of screening were significant predictors for LSIL; and older age and advanced WHO HIV disease stage were significant predictors for HSIL (Supplementary Table 1).

Discussion

In this cross-sectional study conducted among HIV-infected women in Dar es Salaam, Tanzania, we found that the prevalence of cervical abnormalities detected by Pap smear was 8.7%, of which 3.9% was LSIL, 3.4% HSIL and 1.4% ASCUS. Women with older age, lower CD4+cell counts, advanced HIV disease stage and cervical inflammation had a significantly independently higher risk for cervical abnormalities.

There has been a wide range of estimates reported for the prevalence of cervical abnormalities in sub-Saharan Africa in recent years. Ononogbu et al.[15] reported a prevalence estimate of 6% among HIV-infected women in Nigeria. Memiah et al.[16] found that prevalence of cervical lesions was 27% among HIV-infected women in Kenya. Data from South Africa,[17] Uganda,[18] Zambia[19] reported that the prevalence was 66%, 73% and 76%, respectively. This wide range of reported prevalences could be due to true differences in study populations or to differences in cervical screening technique used across studies. For

example, in the study conducted in Uganda, HIV-infected participants were enrolled from a sexually transmitted infection clinic. Study participants may have been motivated to visit the clinic due to the presence of a genital discharge or other symptoms which could be, at least in part, be associated with cervical abnormalities. This might explain the higher observed prevalence of cervical lesions in that study.[18] Some of the studies used monolayer liquid cytology[19] or Visual Inspection with Acetic acid or Lugol's Iodine (VIA/VILI)[16] as the screening technique, and these techniques have been reported to have a higher sensitivity than conventional cytology.[17,20]

The results from previous studies on the association between age and cervical abnormalities among HIV-infected women have been mixed. Several studies have reported that there was no association between age and precancerous cervical lesions,[16,21,22] while others found that age was a significant predictor for cervical abnormalities.[15,19] Ononogbu et al.[15] reported that the risk of screening positive for cervical abnormalities decreased with increasing age, with women aged 40 years and older having lower risk,[15] while Parham et al.[19] found that the relationship between age and the risk cervical lesions was non-linear with women aged 36–40 years at the highest risk. We found that age was positively associated with risk of cervical abnormalities, with older women (≥50 years) having the highest risk. The discrepancy across the available studies in relation to age could be due to residual confounding from sexual behaviours, HPV infection/types and other unmeasured covariates. Age is a likely predictor for increased risk to have a HPV infection and could thereby be associated with cervical abnormalities. Since we did not collect data on HPV status in this study, we were unable to examine the association between age and cervical abnormalities after adjusting for HPV status.

In our study, lower CD4+cell count was associated with a significantly higher risk of cervical abnormalities; this finding is consistent with the results of many previous studies.[15,23–25] HIV infection may increase the risk of cervical abnormalities by permitting HPV persistence and/or by accelerating the development of precancerous cervical lesions in the presence of HPV infection. Several studies have consistently demonstrated that HIV-infected women have a higher rate of persistent HPV infection compared with HIV-negative women[2]; among HIV-infected women, a lower CD4+cell count was significant predictor for high-risk of HPV infection, e.g. HPV genotypes 16 and 18.[26] In addition, it has been suggested that HIV infection was associated with increased risk of HSIL or invasive cervical cancer only among women infected with high-risk HPVs, but not among women without high-risk HPV infection.[27] The interaction between HIV and HPV on the development of cervical cancer deserves further examination, especially in the context of ART. In this study, we did not find any association between ART status and SILs. The lack of association may be due to low power to detect any association, since although 91% of the study participants were on ART, the majority had only been on ART for two months or less.

Our study is not without limitations. First, this is a cross-sectional study, so causal relationships cannot be drawn from the analysis. However, it is unlikely that cervical abnormalities could cause the risk factors studies here. Second, because 91% of the study participants were on ART and most of them initiated within two months from the time of their cervical cancer screen, and because they tended to have lower haemoglobin levels

and CD4+cell counts and be less likely to be pregnant, compared to those who did not receive a Pap smear during the study period (Supplementary Table 2), the study sample may not be representative of HIV-infected women in Dar es Salaam. Thus, it is possible that the risk factors identified are not generalisable to other HIV-infected women populations in sub-Saharan Africa. To address this potential limitation, we accounted for any possible selection bias in the results which could have been induced by a small group of the overall study population having been screened by using inverse probability of selection weighting, where each study participant in the available study population was weighted by her inverse probability of being included in the study as a function of measured covariates. Since the findings from this analysis were similar to those of our primary analysis, we concluded that any possible selection bias had no material impact on the results. However, it should be noted that this method of inverse probability of selection weighting cannot entirely exclude the possibility of selection bias due to unknown and unmeasured confounders.

Despite these limitations, the relatively large sample size in the current study provided us with a reasonable degree of accuracy to estimate the prevalence of cervical abnormalities and to identify its predictors.

In conclusion, in this study, we found that 8.7% of HIV-infected women had cervical SIL by Pap smear screening. Although the number is relatively lower compared to results from some other studies, it still suggests a high prevalence of cervical SIL among Tanzanian HIV-infected women. In addition, we found that lower CD4+cell count and advanced HIV disease stage were associated with a higher risk of cervical SIL. Our results highlight the importance of integrating cervical prevention services, including screening and treatment of SIL, into HIV care and treatment programs in resource-limited settings; in particular, HIV-infected women should receive regular screening for cervical cancer, especially those with CD4+cell counts less than 200 cells/mm^3.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1.

Baseline characteristics of HIV-infected women who received Pap smear screening in HIV care and treatment clinics (*n*=1365).

	Abnormal Pap smear[a]	
	No (*n* = 1246)	**Yes (*n* = 119)**
Age group, years, *n* (%)		
<30	275 (22.0)	17 (14.3)
30–<40	584 (46.9)	57 (47.9)
40–<50	305 (24.2)	27 (22.7)
≥50	82 (6.6)	18 (15.1)
Married, *n* (%)	463 (38.7)	45 (38.8)
Calendar year of screening, *n* (%)		
2006 and 2007	327 (26.3)	43 (36.1)
2008	358 (28.7)	39 (32.8)
2009	561 (45.0)	37 (31.1)
BMI, kg/m^2,*n* (%)		
<18.5	247 (19.8)	27 (22.9)
18.5–<25	691 (55.5)	70 (59.3)
25–<30	220 (17.7)	16 (13.6)
≥30	87 (7.0)	5 (4.2)
Haemoglobin, g/dL, *n* (%)		
<8.5	198 (16.7)	25 (21.9)
8.6–<10	340 (28.6)	30 (26.3)
10—<12	470 (39.5)	30 (39.5)
≥12	181 (15.3	14 (12.3)
CD4 + count, cells/mm^3, *n* (%)		
<50	184 (15.6)	19 (16.4)
50–<100	168 (14.3)	29 (25.0)
100–<200	394 (33.5)	34 (29.3)
≥200	431 (36.6)	34 (29.3)
WHO stage, *n* (%)		
I	105 (8.4)	13 (11.0)
II	270 (21.7)	20 (17.0)
III	759 (61.1)	65 (55.1)
IV	109 (8.8)	20 (16.9)
Current pregnancy, *n* (%)	15(1.2)	1 (0.8)
Months on ART, *n* (%)		
Not on ART	112 (9.0)	11 (9.3)
>0 and <2	928 (74.5)	85 (71.4)
≥2	206 (16.5)	23 (19.3)
Cervical inflammation, n (%)	755 (60.6)	90 (75.6)

BMI: body mass index; ART: antiretroviral therapy; SIL: squamous intraepithelial lesions.

[a]A Pap smear result was defined as abnormal if it was low-grade SIL (LSIL), high-grade SIL (HSIL) or atypical squamous cells of undetermined significance (ASCUS) by the pathologist.

Table 2.

Risk factors associated with abnormal Pap smear results among HIV-infected women.[a]

Variables	*n/N*, %	Univariate PR (95% CI)[b]	*p* value	Multivariate[c] PR (95% CI)	*p* value
Age group, years			0.003		0.001
<30	17/292, 5.8%	0.65 (0.39–1.11)		0.69 (0.41–1.15)	
30–< 40	57/641, 8.9%	1.00		1.00	
40–< 50	27/332, 8.1%	0.91 (0.59–1.42)		0.92 (0.59–1.43)	
≥50	18/100, 18.0%	2.02 (1.24–3.29)		2.36 (1.45–3.84)	
BMI group, kg/m^2			0.09		0.47
<18.5	27/274, 9.9%	1.07 (0.70–1.63)		0.85 (0.55–1.32)	
18.5–< 25	70/761, 9.2%	1.00		1.00	
25–< 30	16/236, 6.8%	0.74 (0.44–1.24)		0.76 (0.45–1.28)	
≥30	5/92, 5.4%	0.59 (0.24–1.43)		0.64 (0.26–1.55)	
WHO stage			0.07		0.12
I and II	33/408, 8.1%	1.00		1.00	
III	65/824, 7.9%	0.98 (0.65–1.46)		0.90 (0.59–1.36)	
IV	20/129, 15.5%	1.92 (1.14–3.22)		1.86 (1.08–3.21)	
CD4+ cell counts, cells/mm^3			<0.001		0.03
<100	47/399, 11.8%	1.61 (1.06–2.45)		1.55 (1.01–2.36)	
100–<200	34/428, 7.9%	1.09 (0.69–1.72)		1.12 (0.71–1.78)	
≥200	34/465, 7.3%	1.00		1.00	
Haemoglobin group, g/dL			0.22		
<8.5	25/223, 11.2%	1.56 (0.84–2.92)			
8.6–<10	30/370, 8.1%	1.13 (0.61–2.08)			
10–<12	45/515, 8.7%	1.22 (0.68–2.17)			
≥12	14/195, 7.2%	1.00			
Pregnancy			0.71		
No	118/1349, 8.8%	1.00			
Yes	1/16, 6.3%	0.71 (0.11–4.80)			
Months on ART			0.28		
Not on ART	11/123, 8.9%	1.00			

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Variables	*n/N*, %	Univariate PR (95% CI)[b]	*p* value	Multivariate[c] PR (95% CI)	*p* value
>0 and <2	85/1013, 8.4%	0.94 (0.52–1.71)			
≥2	23/229, 10.0%	1.12 (0.57–2.23)			
Cervical inflammation[d]			0.001		0.008
No	29/519, 5.6%	1.00		1.00	
Yes	90/845, 10.7%	1.91 (1.27–2.85)		1.73 (1.16–2.60)	
Married			0.99		
No	71/803, 8.8%	1.00			
Yes	45/508, 8.9%	1.00 (0.70–1.43)			
Calendar year of screening			0.002		0.02
2006 or 2007	43/370, 11.6%	1.00		1.00	
2008	39/397, 9.8%	0.85 (0.56–1.27)		0.96 (0.64–1.45)	
2009	37/598, 6.2%	0.53 (0.35–0.81)		0.62 (0.40–0.94)	

PR: prevalence ratio; CI: confidence interval; BMI: body mass index; ART: antiretroviral therapy.

[a]Data analysis based on 1365 cervical screenings from 1365 HIV-infected women.

[b]PR and its 95% confidence from generalised estimating equation (GEE) models using the binomial distribution with log link and exchangeable working correlation structure.

[c]Variables with *p* value <0.2 in univariate analysis were included in the multivariate analysis.

[d]Cervical inflammation was determined through a cytological examination by a pathologist.

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