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Impact of stress and depression on the frequency of squamous intraepithelial lesions

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Abstract

Objective—To explore previously reported associations between cervical squamous lesions and psychological measures of stress and depression.

Methods—In a multicenter cohort study, HIV infected and seronegative comparison women had Pap tests and completed self-report questionnaires including the Perceived Stress Scale-10 (PSS), which measures perceived stress; the PTSD Civilian Symptom Checklist (PCL-C), which measures symptoms of posttraumatic stress disorder; and the Center for Epidemiologic Studies Depression Scale (CES-D), which measures depressive symptoms.

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Results—Median scores were 13 (range 0–38) for the PSS, 24 (range 17–85) for the PCL-C, and 8 (range 0–57), for the CES-D, indicating moderate stress and minimal depression. For PSS, compared to women in the lowest tertile of reported stress, O.R. for SIL was 0.88 (95% C.I. 0.50–1.54) for women in the middle tertile and 0.96 (95% C.I. 0.54–1.68) for women in the highest tertile. For PCL-C, compared to women in the lowest tertile of PTSD symptoms, O.R. for SIL was 0.79 (95% C.I. 0.43–1.41) for women in the middle tertile and 1.17 (95% C.I. 0.68–2.01) for women in the highest tertile. SIL rates were similar for CES-D scores ≥16 (compared to women with lower scores O.R. 1.41, 95% C.I. 0.88–2.26) and ≥ 23 (O.R. 1.39, 95% C.I. 0.81–2.40). In multivariable analysis including number of sexual partners, age, income, ethnicity, and serostatus, stress as measured by PSS and PCL-C, and depressive symptoms as measured by CES-D remained unassociated with SIL.

Conclusions—We found no evidence that stress and depression affect the prevalence of cervical squamous lesions.

Keywords

stress; depression; cervical lesion; Papanicolaou test

Introduction

Abnormal Pap results (squamous intraepithelial lesions, or SILs) are common among women with the human immunodeficiency virus (HIV) (1, 2). Risk factors for abnormal Pap tests include younger age, smoking, number of sexual partners, prior abnormal Pap, and prior cervical disease treatment. However, immune impairment is a crucial factor predicting abnormal cytology. HIV serostatus, CD4 count, and HIV RNA level have been linked to abnormal Pap results, and use of highly active antiretroviral therapy (HAART) may be protective (3).

Stress is a poorly understood potential risk factor for cervical disease. Several authors have suggested that stress increases risk for abnormal Pap results (4–6), and stress management may decrease risk for cervical disease in HIV seropositive women (7). Stress may act on the cervix through modulation of immune function, as the human papillomavirus (HPV), which causes cervical cancer and its precursor, cervical intraepithelial neoplasia (CIN), is often cleared by host immunity (8). The impact of stress on genital immunity is illustrated by its enhancement of herpes reactivation (9) and bacterial vaginosis (10–12). Greater stress has been linked to decrements in measures of cellular immunity in individuals with HIV (13–16), though HAART may ameliorate these effects (17). Stress has also been linked to impaired T-cell response to HPV16, the most virulent genotype of HPV (18). Since cervical cancer prevention requires compliance with screening and treatment protocols, stress may promote the persistence and prevalence of cervical lesions when it interferes with screening and follow-up (19, 20).

However, most studies of the relationship between stress and cervical disease have been small, making it difficult for them to control for potential confounders. We set out to estimate the independent significance of any association between life stress and cytologic findings of squamous intraepithelial lesions (SILs) and to determine the impact of HIV serostatus on that association in a large cohort of women.

Methods

This investigation was part of the Women's Interagency HIV Study (WIHS), a continuing multicenter prospective cohort study of the health of HIV seropositive women and at-risk HIV-uninfected comparison women in the United States. The protocols, recruitment

processes, procedures, and baseline results of the WIHS have been previously described (21). Enrollment began in 1994 at 6 study consortia (Brooklyn, Bronx, Chicago, Los Angeles, San Francisco, and Washington, D.C.) and was expanded to 3,766 women during 2001–2002 to recruit younger women (22). Written informed consent was obtained after local human subjects committees approved. Follow-up continues, but this analysis includes only information obtained between October 1, 2008 and March 30, 2009.

Information on demographics, behavior, and health was obtained via interview every six months, along with a physical examination and Pap test. HIV status was established by Western blot, and for women who seroconverted during study serostatus was assigned according to results at the visit at which Pap test and stress assessments were obtained. Pap tests were interpreted centrally at Dianon (New York, NY, formerly Kyto or Kyto Meridien) according to the 1991 Bethesda system for classification of cervicovaginal cytology (23). All Pap smears were screened by two cytotechnologists blinded to HIV status, with 10% of all negative smears and all abnormal smears reviewed by a cytopathologist. Our outcome variable was a Pap result of SIL or cancer at the study visit coinciding with administration of the stress questionnaires. We excluded 256 women with atypical squamous cells of undetermined significance and 7 with atypical glandular cells.

Participants completed the Perceived Stress Scale-10 (PSS) (24, 25), which measures individuals' perception of stress and coping, the PTSD Civilian Symptom Checklist (PCL-C) (26), and the Center for Epidemiologic Studies Depression Scale (CES-D) (27), which measures depressive symptoms. Scores on the PSS-10 were converted to tertiles for comparison, while women with PCL-C scores over 50 were considered to have post-traumatic stress. Scores on the CES-D above 15 reflect a moderate prevalence of clinical depression (28).

Less than 1% of observations were missing values on covariates; these were inputted using medians for continuous variables and modes for discrete variables. Descriptive comparisons of covariates with stress tertiles were conducted using Pearson's chi-squared test. Univariate and multivariate odds ratios for SIL were determined using logistic regression with Wald confidence intervals. Multivariate logistic models were checked using bootstrapping methods. All analyses were performed in R.

Results

The demographic characteristics of our study group are shown in Table 1. Participants were predominantly of minority ethnicity and of low income. Almost 15% had abnormal Pap test results, although most of these were atypical or low grade.

Median score for the PSS-10 perceived stress measure was 13 (range 0–38) and for the PCL-C was 24 (range 17–85), suggesting moderate stress. As shown in Table 1, stress as measured by PSS was associated with older age, lower income, less education, smoking, and drug use. PSS and PCL-C were correlated ($P < 0.001$ by test for nonzero correlation). Of the 1536 women with completed questionnaires, PCL-C scores were >50, indicating post-traumatic stress, for 149 (10%). Median CES-D score was 8 (range 0–57), suggesting minimal prevalence of depression. CES-D score was also correlated with PSS ($P < 0.001$).

In univariate analysis, SIL was associated with known cervical cancer risk factors, including HIV seropositivity (O.R. 12.62, 95% C.I. 3.96–40.27, $P < 0.001$, compared to seronegative women), ethnicity (O.R. 0.38, 95% C.I. 0.19–0.79, $P = 0.004$ for white and O.R. 0.46, 95% C.I. 0.23–0.95, $P = 0.026$, for other ethnicity compared to African-American), current employment (O.R. 0.28, 95% C.I. 0.15–0.51, $P < 0.001$ for compared to unemployed

women), and current smoking (O.R. 3.24, 95% C.I. 1.71–6.15, P < 0.001, compared to never smokers).

The proportion of women with SIL was not significantly different across stress levels. For PSS, compared to women in the lowest tertile of reported stress, O.R. for SIL was 0.88 (95% C.I. 0.51–.53, P = 0.655) for women in the middle stress tertile and 0.96 (95% C.I. 0.55–1.68, P = 0.886) for women in the highest stress tertile. For PCL-C, compared to women in the lowest tertile of reported PTSD symptoms, O.R. for SIL was 0.78 (95% C.I. 0.44–1.40, P = .415) for women in the middle tertile and 1.17 (95% C.I. 0.69–2.00, P = .560) for women in the highest stress tertile.

Depressive symptoms also were not associated with SIL. SIL rates were similar for CES-D scores above 16 (O.R. 1.41, 95% C.I. 0.88–2.26, P = 0.152) and above 23 (O.R. 1.39, 95% C.I. 0.81–2.40, P = 0.244).

Because prior treatment might have masked significant associations, we repeated analyses to look for a possible correlation between SIL at any time during WIHS and either PSS or PCL-C at the index visit; again no association was found (not shown). Similarly, repeating subset analyses in only the HIV seropositive group did not reveal an association between stress and SIL (not shown). Because HIV-related immunosuppression may have dominated stress as a determinant of SIL, we repeated analyses, limiting assessment only to those women with HIV and CD4 lymphocyte counts >500 and to women without HIV; no association between SIL and PSS score, PCL-C, or CES-D was found. Finally, we found no associations between SIL and combinations of PCL-C and CES-D scores or potentially stressful events including trauma history and self-reported sexual abuse (not shown).

Results of multivariable analysis are shown in Table 2. Odds of SIL were elevated among women with HIV infection and were linked to degree of immunosuppression, race, and current smoking. PSS, PCL-C score >50, and CES-D score >15 were not significantly associated with SIL.

Conclusions

We failed to find an association between prevalent SIL and validated measures of perceived stress, post-traumatic stress, or depressive symptoms, despite previous studies that identified stress as a correlate of abnormal Pap results. Since both stress and SIL are linked to minority ethnicity and measures of socioeconomic status, residual confounding may explain prior findings. Alternatively, we may have failed to identify a link because we had relatively few women with low stress levels. In our high-risk population, other known risks for SIL such as smoking, HIV-related immunosuppression, and multiple sexual partners may dominate any effects of stress. In addition, we assessed stress at a single time point while cervical disease commonly develops over many years, and so we cannot exclude an effect of chronic stress on SIL risk. Finally, our population had been screened and incident cervical cancer precursors treated over many years; stress may have a greater impact on SIL in an unscreened population. However, current stress was not associated with ever having had SIL during our participants' 15-year history of follow-up.

While interventions to minimize life stress may improve quality of life, cervical cancer prevention efforts should remain focused on screening and treatment of precursor lesions. In addition, while stress may not impact SIL risk, it may impair compliance with screening and follow-up among women at risk for cervical cancer. Clinical trials to assess the effect of stress reduction efforts on cervical disease and compliance may improve our understanding of the relationship between stress and cervical cancer risk.

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Table 1

Demographic characteristics of 1536 women completing stress assessments divided into by tertiles of stress score.

	Total		Bottom Tertile (PSS[1] < 8)		Middle Tertile (PSS 8–16)		Top Tertile (PSS ≥ 17)		P-value[2]
Predictor	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	
	1536		446		553		537		
Age									.024
<30	91	5.9	29	6.5	40	7.2	22	4.1	
30–40	367	23.9	119	26.7	140	25.3	108	20.1	
40–50	617	40.1	175	39.2	210	38.0	232	43.2	
50–60	380	24.7	98	22.0	133	24.1	149	27.7	
60–70	68	4.4	18	4.0	28	5.1	22	4.1	
>70	13	0.8	7	1.6	2	0.4	4	0.7	
Race/ethnicity									.595
White	332	21.6	94	21.1	129	23.3	108	20.1	
African-American	925	60.1	275	61.7	328	59.3	321	59.8	
Hispanic	225	14.6	64	14.3	73	13.2	88	16.4	
Other	56	3.6	13	2.9	23	4.2	20	3.7	
HIV Status									.592
HIV+	1088	70.9	309	69.3	391	70.7	388	72.3	
HIV−	448	29.1	137	30.7	162	29.3	149	27.7	
Annual income									<.001
< $6,000	255	16.6	73	16.4	90	16.3	92	17.1	
$6,000–$12,000	493	32.1	115	25.8	169	30.6	209	38.9	
$12,000–$18,000	206	13.4	57	12.8	69	12.5	80	14.9	
$18,000–$24,000	133	8.6	48	10.8	44	8.0	41	7.6	
$24,000–$30,000	103	6.7	31	7.0	44	8.0	28	5.2	
$30,000–$36,000	79	5.1	27	6.1	27	4.9	25	4.7	
$36,000–$75,000	184	12.0	67	15.0	75	13.6	42	7.8	
>$75,000	78	5.1	26	5.8	34	6.1	18	3.4	
Missing	5	0.3	2	0.4	1	0.2	2	0.4	

	Total		Bottom Tertile (PSS[1] < 8)		Middle Tertile (PSS 8–16)		Top Tertile (PSS ≥ 17)		P-value[2]
Predictor	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	
Education									.003
No Schooling	7	0.5	0	0.0	5	0.9	2	0.4	
Grades 1–6	70	4.6	18	4.0	23	4.2	29	5.4	
Grades 7–11	523	34.1	154	34.5	168	30.4	201	37.4	
Completed HS	453	29.5	112	25.1	171	30.9	170	31.7	
Some College	386	25.1	128	28.7	154	27.8	104	19.4	
Completed College	67	4.4	22	4.9	22	4.0	23	4.3	
Attended Grad School	27	1.8	12	2.7	10	1.8	5	0.9	
Missing	3	0.2	0	0.0	0	0.0	3	0.6	
Condom Use									.393
Always / NA[3]	1071	69.7	301	67.5	386	69.8	384	71.5	
Sometimes/never	465	20.3	145	32.5	167	30.2	153	28.5	
Tobacco use within 6 months									<.001
Yes	650	42.4	162	36.3	215	38.9	273	50.8	
No	886	57.7	284	63.7	338	61.1	264	49.2	
Intravenous drug use									.001
Yes	17	1.1	1	0.2	3	0.5	13	2.4	
No	1518	98.8	444	99.6	550	99.5	524	97.6	
Missing	1	0.1	1	0.2	0	0.0	0	0.0	
Pap Result									
Normal	1229	80.0	362	81.2	442	79.9	425	79.1	0.909[4]
ASC-US[5]	228	14.8	59	13.2	84	15.2	85	15.8	
ASC-HGSIL[6]	2	0.1	1	0.2	1	0.2	0	0.0	
Lgsil[7]	63	4.1	22	4.9	18	3.3	23	4.3	
HGSIL/CIN2[8]	9	0.6	1	0.2	7	1.3	1	0.2	
HGSIL/CIN3[9]	4	0.3	1	0.2	1	0.2	2	0.4	
Carcinoma	1	0.1	0	0.0	0	0.0	1	0.2	

	Total		Bottom Tertile (PSS[1] < 8)		Middle Tertile (PSS 8–16)		Top Tertile (PSS ≥ 17)		P-value[2]
Predictor	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	
CD4 (HIV+ Only)									.503
<200	137	12.7	34	11.1	47	12.1	56	14.6	
200–500	406	37.7	115	37.7	142	36.4	149	38.9	
≥500	535	49.6	156	51.1	201	51.5	178	46.5	

[1] Perceived Stress Score

[2] By chi-square test

[3] Not sexually active

[4] Compares Normal vs. all others, excluding ASC-US.

[5] Atypical squamous cells of undetermined significance (excluded from analyses).

[6] Atypical squamous cells, cannot exclude high grade lesion

[7] Low grade squamous intraepithelial lesion

[8] High grade squamous intraepithelial lesion, moderate dysplasia

[9] High grade squamous intraepithelial lesion, severe dysplasia

Table 2

Multivariable analysis of associations of demographic and biological risk factors for squamous intraepithelial lesions on Pap testing.

Predictor	Adjusted Odds Ratio	95% Confidence Interval	p
Serostatus			
HIV−	1.00		
HIV+, CD4 >500	3.08	2.3–4.0	<.001
HIV+, CD4 200–500	9.47	5.5–16.3	<.001
HIV+, CD4 < 200	29.16	19.2–44.4	<.001
Race			
African American	1.00		
Whites and other race	0.44	0.25–0.79	.006
Income			
>$12,000/yr	1.00		
≤ $12,000/yr	0.80	0.47–1.35	.40
Tobacco use past six months			
No	1.00		
Yes	3.67	2.1–6.3	<.001
Number of sexual partners past six months, per partner	0.77	0.50–1.09	.12
Age in years			
<40	1.00		
40–50	0.65	0.35–1.21	0.18
>50	0.61	0.31–1.22	0.16
Perceived stress (PSS)[1]			
Score <8	1.00		
Score 8–16	0.77	0.43–1.40	0.40
Score ≥ 17	0.67	0.37–1.24	0.21
Post traumatic stress (PCL-C)[2]			
No (score ≤50)	1.00		
Yes (score >50)	0.58	0.23–1.45	.25
Depressive symptoms (CES-D)[3]			
No (score <16)	1.00		
Yes (score ≥ 16)	1.10	0.66–1.84	.72