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Article

HIV and cervical cancer in Kenya

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Abstract

Objectives: To determine the effect of the HIV epidemic on invasive cervical cancer in Kenya. *Methods:* Of the 3902 women who were diagnosed with reproductive tract malignancies at Kenyatta National Hospital (KNH) from 1989 to 1998, 85% had invasive cervical cancer. Age at presentation and severity of cervical cancer were studied for a 9-year period when national HIV prevalence went from 5% to 5–10%, to 10–15%. *Results:* There was no significant change in either age at presentation or severity of cervical cancer. Of the 118 (5%) women who were tested for HIV, 36 (31%) were seropositive. These women were 5 years younger at presentation than HIV-negative women. *Conclusions:* A two- to three-fold increase in HIV prevalence in Kenya did not seem to have a proportional effect on the incidence of cervical cancer. Yet, HIV-positive women who presented with cervical cancer were significantly younger than HIV-negative women.

Keywords: HIV; Cervical cancer; Kenya

1. Introduction

Cervical cancer is one of the AIDS-associated or AIDS-defining illnesses [1,2]. This is based on the observation in the early 1990s that women infected with HIV had a significantly higher risk of developing squamous intraepithelial lesions (SIL), the pre-malignant stage of cervical cancer [1,2]. Since then, studies have pointed to an association between HIV infection and both invasive carcinoma of the cervix and a faster progression to more advanced stages of cervical carcinoma, the latter with higher treatment failures and more recurrences [3–6]. Other cancers associated with

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HIV/AIDS include Kaposi sarcoma, non-Hodgkin's lymphoma (NHL), and childhood leiomyosarcoma [7–9].

Other studies have suggested a significant association between pre-malignant cervical lesions and HIV infection [10–12], and the association of HIV infection and cervical dysplasia has been confirmed [12]. Yet, the correlation between HIV infection and invasive cervical cancer remains inconclusive [3,6,13]. Reports on iatrogenically-induced immunosuppression to prevent rejection of transplanted organs clearly show an increased risk of cervical dysplasia and cervical cancer, however, suggesting a relationship between immunosuppression and cervical cancer [14,15].

If HIV infection increases both the likelihood of cervical cancer developing from dysplasia and the likelihood of a worsening of the stage of cervical cancer, a correlation could be expected between the rising HIV prevalence in Kenya and the incidence of cervical cancer.

This retrospective study was undertaken to examine the effects of increasing HIV prevalence in Kenya on patient age at presentation, and on the incidence and severity of invasive cervical cancer.

2. Materials and methods

Most patients treated for reproductive-tract cancers in the Radiotherapy Department of Kenyatta National Hospital (KNH), Nairobi, Kenya, are referred by the hospital's Department of Obstetrics and Gynecology. Others are referred from other hospitals. There are three cervical cancer treatment centers in Kenya: Kenyatta National Hospital (public) and Nairobi Hospital (private) in Nairobi, and Nyanza Provincial General Hospital (public) in the western part of the country.

A private hospital, Nairobi Hospital is not financially accessible to the majority of the Kenyan population. Nyanza Provincial General Hospital is not very operational because of a lack of qualified staff and adequate equipment. KNH therefore remains the national cervical cancer treatment center. It functions as a teaching as well as a referral hospital.

The number of patients admitted at Kenyatta National Hospital and the number of women with reproductive-tract cancer were obtained from the hospital's annual reports. All women diagnosed with reproductive-tract cancers in the departments of Obstetrics and Gynecology and Radiotherapy were included. Case records of patients with cervical cancer between 1989 and 1998 were retrieved from the Records Department at KNH.

At KNH, records not in use are usually destroyed after 10 years. All available records of patients with a diagnosis of reproductive-tract malignancies were assessed. Cases with a histologically-verified diagnosis of invasive cervical cancer were included. Cases with clinical diagnosis of invasive cancer but without histological results were reviewed by a gynecologist (P.G.) and were classified as either probable cases or unlikely cases of invasive cervical cancer. Probable cases of invasive cervical cancer were defined as cases with both a history suggestive of cervical cancer and a clinical examination highly suggestive of cervical cancer, but without histological confirmation. Unlikely cases of invasive cervical cancer were defined as cases with a history suggestive of invasive cervical cancer but an inconclusive clinical examination in the absence of histological results. Sociodemographic characteristics, number of pregnancies, history of Pap-smear testing, HIV serostatus, and International Federation of Gynecology and Obstetrics (FIGO) staging of cervical cancer — including histological subtypes and differentiations — were obtained from the case records of women with reproductive-tract malignancies.

2.1. Statistical methods

Data generated from the case records were coded and completed the questionnaire. They were then processed with statistical package SPSS, version 9.0 (SPSS Inc. Chicago, IL, USA). Analysis was stratified into three periods, 1989–90, 1991–94, and 1995–98, with respective HIV

Table 1
Distribution of female reproductive-tract cancers at KNH, 1989–1998

Site of malignancy	1989		1991		1992		1994		1995		1996		1997		1998	
	N	%	*N*	%	*N*	%	*N*	%	*N*	%	*N*	%	*N*	%	*N*	%
Cervix	401	84.7	398	85.5	378	92.8	210	86.4	313	85	370	84.2	267	83.1	343	81.8
Uterus	6	1.2	12	2.5	12	2.9	4	1.6	12	3.2	9	2.1	9	2.8	10	2.3
Ovary	54	11.4	38	8.1			24	9.8	26	7.1	46	10.4	29	9.0	45	10.7
Placenta			6	1.2	8	1.9	3	1.2	2	0.5	4	0.9	6	1.8	8	1.9
Vagina/vulva[a]													8	2.4	13	0.3
unspecified	12	2.5	11	2.3	9	2.2	2	0.8	16	4.3	10	2.2	2	0.6	0	
Total new female cancers	473		465		407		243		368		439		321		419	
New gynecology patients	1800		2218		1095		609		1082		1444		1443		1790	

Data for 1990 and 1993 were incomplete. The table based on hospital reports.
[a] Coding for disease processes changed in 1997 from International Classification of Diseases 9 (ICD 9) to ICD 10.

prevalence of 5%, 5–10%, and 10–15% in women of reproductive age [16]. The number of cervical cancer cases, age at presentation, and the severity of cervical cancer were compared with HIV prevalence rates for women, using Yates corrected Chi-square test. Differences mean ages were tested using Student's *t*-test.

3. Results

3.1. Magnitude of invasive cervical cancer

Of the 3902 women who were diagnosed with reproductive-tract malignancies at Kenyatta National Hospital (KNH) from 1989 to 1998, 3327 (85%) had invasive cervical cancer. Seventy six percent (2542/3327) of the case records reporting cervical cancer were retrieved for review, and 2382 (94%) of those were indicative of invasive cervical cancer. The other case records (26%) could not be found because of misfiling or loss.

As shown in Table 1, the number of cervical cancer cases and the number of new gynecology cases did not change significantly ($P > 0.05$) over an 8-year period (data for 1990 and 1993 were missing). Similarly, there was no significant change in proportion between cervical and other female reproductive-tract cancers between 1989 and 1998 (84% vs. 82%, $P = 0.458$).

3.2. Demographic characteristics of women

The mean age at diagnosis of cervical cancer was 47 ± 12 years, with approximately 30% of the women between 40 and 49 years. The mean age at presentation varied from 45 ± 13 years in 1989 to 47 ± 13 years in 1998 ($P = 0.256$). As shown in Table 2, the proportion of women with cervical cancer below 35 years did not change significantly over this period. Overall, women in Stage 1 to Stage IIa were 3 years younger than those in Stage IIb to Stage IV (45 ± 12 years vs. $48 + 12$ years, $P < 0.001$).

The 26% of women with secondary-school education or higher (51/195) were more likely to present with Stage I to Stage IIa than the 14% with lower education (240/1662, $P < 0.001$).

Among women with invasive cervical cancer, there was a significant decrease in the proportion of women with high parity (parity ≥ 5), from 74% in 1989–90 to 68% in 1995–98, ($P = 0.002$) (Table 2). Nulliparity was rare in this group (2%). Most patients were 'grandes multiparae' (72%). In addition, high-parity women were significantly more likely to have Stage IIb–IV than women of lower

Table 2
Demographic characteristics of patients with invasive cervical cancer reviewed in 1989–1990, 1991–1994 and 1995–1998, in relation to women's HIV seroprevalence rates in Kenya

Characteristics(s)	1989–1990		1991–1994		1995–1998	
	N	%	*N*	%	*N*	%
HIV positive	6/23	26.1	12/34	35.2	18/61	29.5
Mean age ± S.D. (years)	47 ± 13		47 ± 12		47 ± 13	
Age < 35 years	(436)	21	(950)	17	(970)	19
Marital status	*N* = 436		*N* = 950		*N* = 970	
Married	331	75.9	697	73.3	671	69.1
Single	19	4.3	54	5.6	87	8.9
Other	86	19.7	199	20.9	212	21.8
Previous Pap test	19/271	7	31/827	3.7	52/734	7.1
Parity						
Nullipara	9/437	2.1	14/921	1.5	18/960	1.8
Para 1–4	103/437	23.5	211/921	22.9	283/960	29.4
Para 5 +	325/437	74.3	696/921	75.5	659/960	68.6

S.D., standard deviation.

Table 3
Clinical (FIGO) staging of invasive cervical cancer over time

Stage of CA CX	1989 $N = 130$	1990 $N = 319$	1991 $N = 257$	1992 $N = 251$	1993 $N = 236$	1994 $N = 213$	1995 $N = 231$	1996 $N = 229$	1997 $N = 218$	1998 $N = 298$
Stage I	4%	9%	8%	6%	6%	8%	5%	6%	2%	4%
A	0	1	0	0	1	1	1	0	0	0
B	5	26	21	16	12	15	11	13	4	11
Stage II	45%	34%	34%	35%	39%	30%	33%	41%	28%	32%
A	11	23	19	23	16	11	20	32	15	19
B	47	85	68	64	77	53	57	61	46	75
Stage III	32%	42%	45%	44%	42%	42%	39%	39%	42%	36%
A	8	28	16	18	23	21	26	13	15	33
B	34	105	100	92	76	68	63	76	76	75
Stage IV	13%	7%	6%	10%	8%	11%	12%	9%	14%	9%
A	7	13	9	7	7	14	19	12	10	20
B	10	10	6	17	11	10	9	9	20	8
Unspecified stage	6% 8	9% 28	7% 18	6% 14	6% 13	9% 20	11% 25	6% 13	15% 32	19% 57

Abbreviations: CA CX, invasive cervical cancer; *N*, total case records reviewed.

parity (86% vs. 82%, $P = 0.024$). Approximately 77% (1832/2382) of all case records showed Pap-smear history, and only 6% of them (102/1832) indicated that a Pap-smear had ever been done.

Of the 2382 patients with invasive cervical cancer, 118 (5%) had been tested for HIV, 36 of whom (31%) were HIV seropositive. The number of women tested for HIV did not differ between the periods 1989–90, 1991–94, and 1995–98 (Table 2). Women tested for HIV were significantly younger (42 ± 11 vs. 47 ± 12 years, $P < 0.001$). HIV-positive women were 5 years younger than HIV-negative women at the time of invasive cer-

Table 4
Clinical and histological classification of invasive cervical cancer according to HIV seroprevalence rates in women

FIGO clinical stage	1989–1990		1991–1994		1995–1998		*P*-value for trend
	$N = 413$	%	$N = 892$	%	$N = 849$	%	
Stage I	32	7.7	6	7.3	40	4.7	0.004
Stage II	166	40.1	331	37.1	325	38.2	0.091
Stage III	175	42.3	414	46.4	377	44.4	0.032
Stage IV	40	9.6	81	9.1	107	12.6	0.357
Stage I–IIa	66	15.9	135	15.1	126	14.8	0.880
Stage IIb–IV	347	84.0	757	84.8	723	85.1	
Histological type	$N = 332$		$N = 721$		$N = 719$		
Squamous cell carcinoma	274	82.3	619	85.8	637	88.5	0.026
Adenocarcinoma	17	5.1	36	4.9	41	5.7	0.685
Adenosquamous	1	0.3	4	0.5	2	0.2	–
Anaplastic	40	12	62	8.5	38	5.2	< 0.001
Differentiation of squamous cell carcinoma	$N = 159$		$N = 425$		$N = 435$		
Well	18	11.3	59	13.8	63	14.4	0.018
Moderate	49	30.8	127	29.8	133	30.5	0.957
Poor	90	56.6	239	56.2	239	54.9	0.154

vical cancer diagnosis (38 ± 8 vs. 43 ± 12 years, $P = 0.015$).

3.3. Severity of invasive cervical cancer: clinical (FIGO) staging

Ninety percent of the cases of invasive cervical cancer were clinically staged, of which 6.4% were Stage I, 38.2% Stage II, 44.8% Stage III, and 10.6% Stage IV. Stage I accounted for less than 10% of the cases for any given year, with a range of 2–9% (Table 3). The proportion of women from Stage I to Stage IIa (13% in 1989 vs. 12% in 1998), or Stage IIb to Stage IV (87% in 1989 vs. 88% in 1998), did not change over time.

3.4. Cervical cancer histological subtypes

Of the 74% (1772/2382) histologically verified cases of invasive cervical cancer, squamous cell carcinoma was the most common histological subtype (1530/1772, 86%). Adenosquamous cancer was rare (7/1772, 0.4%), while adenocarcinoma represented 5% (95/1772) and anaplastic cervical cancer 8% (140/1772) of all invasive cervical cancers. The proportion of squamous cell carcinoma significantly increased from 83% in 1989–90 to 89% in 1995–98 ($P = 0.026$), with a concurrent decrease (from 12% to 5%, $P < 0.001$) in the proportion of cases reported as anaplastic (Table 4).

Histological differentiation was reported in 69% (1223/1772) of all histologically verified cases. Overall, approximately 48% of the histological subtypes were reported as poorly differentiated, 28% as moderately-well differentiated, 13% as well-differentiated and 11% as anaplastic. As shown in Table 4, cases of well-differentiated squamous cell carcinoma increased from 6% in 1989–90 to 15% in 1995–98 while moderately and poorly differentiated types did not differ over the same periods.

4. Discussion

Kenya is one of the sub-Saharan countries ravaged by the HIV/AIDS epidemic. By 1999, it was estimated that two million Kenyans were infected with the HIV virus [6]. In 1998, the prevalence rate of HIV infection among adults in Kenya was 15% [6]. Of the two million cases, one million were female (ratio of men/women in Kenya is 1:1).

Several cancers have been designated as AIDS-associated, with a significantly increased risk among HIV seropositive individuals [1,2,7,9]. Cervical cancer is gradually recognized as a sexually transmitted disease. The sexually transmitted etiological agent has been identified as the human papillomavirus (HPV). Women with HIV infection are more likely to have a concurrent HPV infection [11,17]. There is firm evidence, however, that HIV is independently associated with an increased risk for cervical intra-epithelial neoplasm [10–12]. Some studies suggest that HIV infection is associated with the rapid progression of HPV-related cervical pre-malignant lesions to invasive cervical cancer or to advanced invasive cervical cancer [4,18].

In Kenya, the incidence of cervical cancer was estimated at 45/100 000 in the early 1980s. Over the next 10 years, the proportional incidence of invasive cervical cancer among new gynecology patients or among women with reproductive-tract cancer did not significantly change. HIV prevalence, however, increased two- to three-fold over the same period in women of reproductive age. These data should be interpreted with caution, as many confounders may have masked the relationship of cervical cancer and HIV. Some of the confounders could include different referral patterns and vertical intervention programs such as screening for cervical cancer. Yet, there is no evidence of change in the referral system for that period in Kenya, and no intervention program that could have influenced the number of reported or treated cases. Cost sharing (user fees), which was introduced in 1989, did not significantly decrease the number of patients' visits at KNH.

In Uganda, Parkin et al. [9] reported a marked increase in the incidence of cervical cancer since 1960, with stabilization in 1990. In Harare,

Chokunonga et al. [7] did not find any increase in the incidence of cervical cancer over a 6-year period, from 1990 to 1995. Data from a teaching hospital in Lusaka, Zambia [19], showed no change in the frequency of cervical cancer during the 1980s. Goedert et al. [20] found no increase in the incidence of invasive cervical cancer among US women and women from Puerto Rico, whereas Rabkin et al. [21] documented a decrease in the incidence of cervical cancer among US black women between 1970 and 1988.

Unlike cervical cancer, the incidence of Kaposi sarcoma seems to mirror the incidence/prevalence of HIV [7,9]. Kaposi sarcoma incidence significantly increased in HIV-infected individuals [20].

If HIV infection indeed shortens the latent period observed in the progression from pre-malignant cervical lesions to invasive disease, then women with HIV infection and invasive cervical cancer are expected to be younger than women with invasive cervical cancer only. Moreover, as HIV infection is much more common among young people, one could expect an earlier onset of the process of cervical cancer in this group. In Kenya, the peak age for HIV infection among women is 25–29 years. Our retrospective study shows that, over the 9 years considered, the mean age of women presenting with invasive cervical cancer remained stable at 47 years whereas the prevalence of HIV in women increased two- to three-fold. Similar observations have been reported in Zimbabwe [7] and Uganda [9] cancer-registry studies. The apparent absence of change in age of presentation could be due to several factors. One hypothesis is that HIV-infected women may die from HIV-related opportunistic infections before they develop invasive cervical cancer. The mean survival time for women with HIV infection in Kenya is 5 years [16].

Our study did show that among women tested for HIV infection, HIV-seropositive women with invasive cervical cancer were 5 years younger than HIV-negative women with invasive cervical cancer. The number of women tested for HIV in our study was low (5%), and they were significantly younger than those who were not tested, suggesting a selection bias in HIV testing.

HIV testing is not a standard protocol at KNH. Because of the nature of our study design, it was not possible to know why tests were done. Our findings, however, are similar to those of Lomalisa et al. [22] who reported that HIV-seropositive women in South Africa presented with invasive cervical cancer 10 years earlier than HIV-negative women. Among the 5% women tested for HIV in our study, 31% were HIV positive. This is higher than the 2.9% reported by Rogo and Kavoo [23] in Kenya, the 7.5% reported by Lomalisa et al. [22], and the 12.6% reported by Sitas et al. in South Africa [6] among women with invasive cervical cancer.

The FIGO clinical staging and histological subtypes were used to assess the severity of cervical cancer. Our finding 6.4% of the women in Stage I is close to the 7% reported by Rogo et al. in 1990 using data from 1968 to 1979 from the same hospital [24]. The proportion of poorly-differentiated subtypes to well-differentiated cervical cancers did not significantly change over time. Our data show no change in the severity of cervical cancer over time at KNH, suggesting a lack of correlation between HIV prevalence in women and severity of cervical cancer. This observation is to be taken with caution as the HIV status of the women was unknown in most cases. Comparing time periods with different HIV infection prevalence, or with no HIV infection, may therefore be a very crude indicator.

The lack of correlation between HIV infection prevalence in women and mean age of presentation, proportional incidence of cervical cancer and severity of cervical cancer could be due to different reasons. HIV infection among women with cervical cancer may be an indicator of risky sexual behavior or shared risk factors. The fate of untreated HIV-related pre-malignant cervical lesions is unknown. One hypothesis is that HIV-infected women probably die before they develop invasive cervical cancer. To date, this hypothesis has not been proven in a thorough clinical study in Kenya. The lack of correlation is most likely due to prevalent opportunistic infections such as tuberculosis; a result of the unavailability of antiretroviral therapy, these infections may shorten the survival time of HIV-positive women. It is

also possible that the number of HIV-infected women with invasive cervical cancer may be too low to influence the mean age of presentation and the total number of cervical cancer cases.

In conclusion, despite a probable selection bias in HIV testing, our study suggests that HIV-seropositive women with cervical cancer present significantly younger than HIV-negative patients. This study did not show an increase in the proportion of invasive cervical cancer among the reproductive-tract cancers of women, nor did it show changes in mean age of presentation or severity of invasive cervical cancer over time. HIV prevalence has increased over time, however, doubling and tripling among women of reproductive age. Further studies are needed to determine the relationship of HIV infection and invasive cervical cancer.

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